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CEREBRAL CIRCULATION

XXXII. EFFECT OF STIMULATION OF THE SYMPATHETIC NERVE ON THE PIAL VESSELS IN THE ISOLATED HEAD

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In studying the circulation of the brain by direct observation of the pial vessels, Wolff and one of us (H. S. F.),[1] Cobb[2] and others have presented evidence that constriction of the cerebral arteries in the intact animal may be accomplished by stimulation of the ipsilateral cervical sympathetic nerve. Histologic studies by Chorobski and Penfield,[3] moreover, suggest that there are efferent nerve fibers, originating in the cervical sympathetic trunk, which supply the ipsilateral arteries of the pia mater and the brain. Finesinger and Putnam[4] added support to these results by experiments on partially isolated heads perfused mechanically with the animal's own blood.

In these perfusion experiments, however, owing to the preservation of the spinal cord and its vascular supply, the possibility of hormonal vasoconstriction (by reflex excitation of the suprarenal glands, for example, through the intact cord) cannot be discounted. A hormone so released, in fact, could conceivably reach and affect the cerebral

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1. Forbes, H. S., and Wolff, H. G.: Cerebral Circulation: III. The Vasomotor Control of Cerebral Vessels, Arch. Neurol. & Psychiat. **19**:1057 (June) 1928.

2. Cobb, S.: Cerebral Circulation: IX. The Relationship of the Cervical Sympathetic Nerve to Cerebral Blood Supply, Am. J. M. Sc. **178**:528, 1929. Cobb, S., and Finesinger, J. E.: Cerebral Circulation: XIX. The Vagal Pathway of the Vasodilator Impulses, Arch. Neurol. & Psychiat. **28**:1243 (Dec.) 1932.

3. Chorobski, J., and Penfield, W.: Cerebral Vasodilator Nerves and Their Pathway From the Medulla Oblongata, Arch. Neurol. & Psychiat. **28**:1257 (Dec.) 1932.

4. Finesinger, J. E., and Putnam, T. J.: Cerebral Circulation: XXII. Induced Variations in Volume Flow Through the Brain Perfused at Constant Pressure, Arch. Neurol. & Psychiat. **30**:775 (Oct.) 1933.

vessels either through the perfusing blood or through the circulation of the cord.

While it is apparent, therefore, that stimulation of the cervical sympathetic nerve is followed by constriction of the ipsilateral pial arteries in the intact animal or in the partially isolated head, it was felt desirable to devise a crucial experiment in which a closer approach to physiologic conditions prevailed, at the same time eliminating all effects other than the direct action of stimulation of the sympathetic nerve on the intracranial vessels. Therefore, direct observations of the pial vessels in a head completely isolated from the body were made, the circulation of the isolated head being maintained by direct arterial and venous anastomosis with a second, intact animal serving as donor. It was thought that while completely negative results might not disprove the theory of direct "sympathetic vasoconstriction," positive results would strongly fortify the existing evidence.

TECHNIC

Healthy cats weighing between 3 and 5 Kg. each were selected for all experiments, the donor and the recipient receiving the same anesthetic, either 7 cc. of 1 per cent amytal or 0.6 cc. of dial per kilogram of body weight, intraperitoneally. A cranial window [5] permitting direct microscopic observation and measurement of the pial vessels was inserted into the skull of the recipient's head. The donor was provided with a cannula (carotid or femoral artery) for recording the systemic blood pressure in millimeters of mercury. An intratracheal cannula was introduced in case the necessity for artificial respiration arose. Care was taken to maintain the normal body temperature throughout each experiment by means of electric heating mats.

Circulation in the recipient's head was effected by direct anastomosis of the cervical vessels of each animal. In the earlier experiments right-angled glass cannulas were inserted into the vessels of the respective animals and connected with rubber tubing. As this method entailed the intravenous administration of heparin (from 70 to 80 mg. per kilogram) into each animal, a simpler plan was resorted to, consisting of direct vessel-to-vessel (endothelium-to-endothelium) anastomosis by means of Crile's cannulas. While the cannulas were being inserted, the vessels were clamped with rubber-covered serrefines.

Anastomosis was always made between the cardiac portion of the donor's common carotid artery and the cephalic end of the recipient's common carotid artery. Venous connections were made in a like manner, one carotid-carotid and one jugular-jugular anastomosis sufficing to maintain adequate circulation. The artery supplying the observed hemisphere of the isolated head served as the recipient vessel. While the vessels were shut off during preparation for anastomosis, a satisfactory flow of blood in the recipient's head was preserved (until isolation was completed) via the vertebral, spinal and opposite carotid arteries.

Dissection of the cervical sympathetic and vagus nerves on the observed side of the isolated head was carried out next, with extreme care to avoid traction.

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5. Forbes, H. S.: Cerebral Circulation: I. Observations and Measurements of Pial Vessels, Arch. Neurol. & Psychiat. **19**:751 (May) 1928.

The nerves were then tied and cut in readiness for stimulation. This was accomplished by means of either unipolar or bipolar electrodes attached to a Harvard inductorium supplied by one dry battery. The strength of the current was maintained at relatively physiologic levels by adjusting the coil at distances of from 8 to 12 cm.

Isolation of the recipient's head was effected by tightly clamping the animal's neck with a simple hinged iron clamp provided with a bolt and thumbscrew at the end opposite to the hinge, a method which proved capable of effectively crushing the neck, vertebrae and cord, so that leakage of neither blood nor spinal fluid ensued. The animal's body died of asphyxia shortly after the neck was clamped, permitting complete separation and removal of the head peripheral to the clamp.

Cross-circulation from the donor to the recipient's head was always established just prior to tightening the clamp on the neck (by removing the sereffines), thus avoiding any interruption of flow through the vessels of the isolated head.

On clamping the neck, the cerebrospinal fluid pressure of the recipient generally became so greatly elevated that free escape of spinal fluid through the cisternal needle was allowed in order to decrease the undue tension. In two experiments the cerebrospinal fluid pressure of each head, in millimeters of Ringer's solution, was obtained by cisternal puncture.

Cross-circulation having been established, direct microscopic measurement of the pial vessels in the isolated head could be made, together with records of the systemic blood pressure of the donor, the cerebrospinal fluid pressures of each animal and changes in the pupils (ipsilateral dilatation following stimulation of the sympathetic nerve). Circulation was considered adequate when the flow of arterial blood was not visibly retarded and when the color of the arterial and the venous blood did not appear abnormal. Before each experimental procedure, readings of the diameter of the observed artery were recorded for at least two (usually five or more) minutes, no procedure being carried out if the size of the vessel varied during this preliminary period or if the blood pressure of the donor changed significantly.

Control procedures consisted in stimulation of other nerves (the contralateral sympathetic and both vagus nerves of the recipient as well as the donor's cervical sympathetic and vagus nerves). A summary of results in tabular form is shown in the table, an individual chart in figure 1, and photomicrographs of a typical vasoconstriction in figures 2 and 3.

RESULTS AND COMMENT

1. Stimulation of the cervical sympathetic nerve was performed in a series of ten completely isolated heads. In all experiments save one vasoconstriction occurred one or more times. Failure of response to stimulation almost invariably occurred in earlier experiments before the technic was perfected, and could usually be attributed to impairment of the circulation due to an excessively low blood pressure in the donor, to partial thrombosis of the vessels supplying the isolated head or to the presence of many intravascular emboli. It should be stated also that occasionally vasoconstriction was obtained even in the face of one or more of these complications. The single preparation rendering completely negative results was marked throughout by an inadequate flow

of blood. In other words, when favorable circulatory conditions prevailed, positive results were invariably attained.

It is worthy of emphasis that the pial arteries exhibited no marked vasoconstriction similar to that which occurs in the vessels of the skin (ear) on stimulation of the sympathetic nerve, when the diameter of an artery may decrease 80 per cent or more of its original size, some-

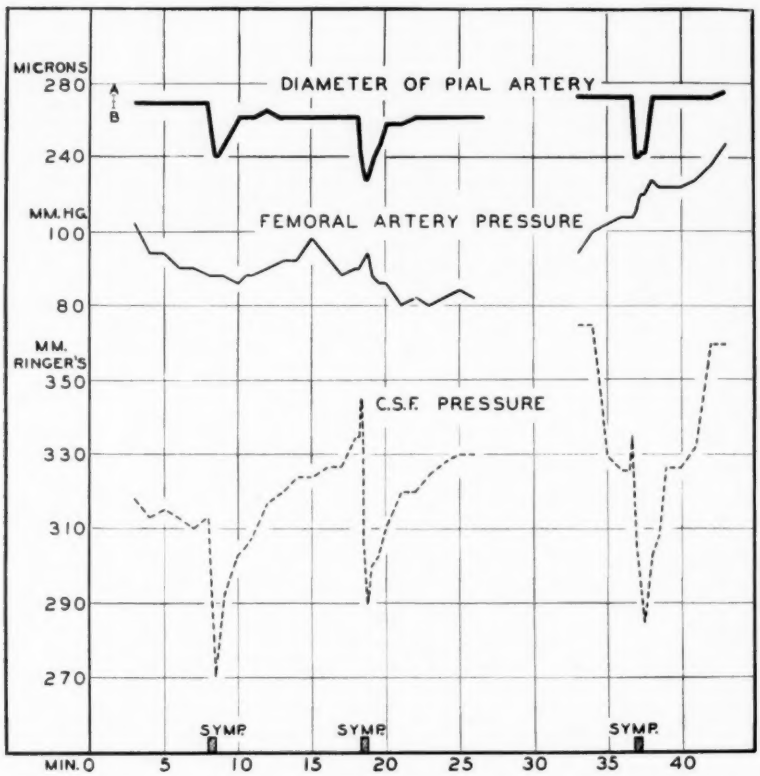

Fig. 1.—Chart showing the results of stimulation of the sympathetic nerve in the isolated head. The sympathetic nerve was stimulated at intervals as indicated by the cross-hatched areas labeled "*symp.*" along the abscissa. The stimulation was made with a faradic current (Harvard inductorium; coil distance, 12 cm.) applied with a unipolar electrode to the nerve trunk. The ordinate represents the diameter of the pial artery in the isolated head (left parietal region) in microns, the femoral artery pressure of the donor in millimeters of mercury and the cerebrospinal fluid pressure of the isolated head in millimeters of Ringer's solution. The abscissa represents time in minutes. (Records were made at intervals of one minute or less.) The broad line, which indicates the diameter of the pial artery, is so plotted that its upper edge records the correct measurements and time relations. The short line *A-B* represents the smallest degree of change in the diameter which could be measured accurately. A change of half this extent is of doubtful validity.

times being almost completely obliterated.[6] The pial vessels, in other words, showed no changes on stimulation of the sympathetic nerve that could be construed in any sense as vascular "spasm."

The table shows that the degree of vasoconstriction ranged from 2.5 to 17 per cent, with an average for all the positive trials of 10 per cent.

Stimulation of the contralateral cervical sympathetic nerve failed at all times to evoke a response affecting the diameter of the observed

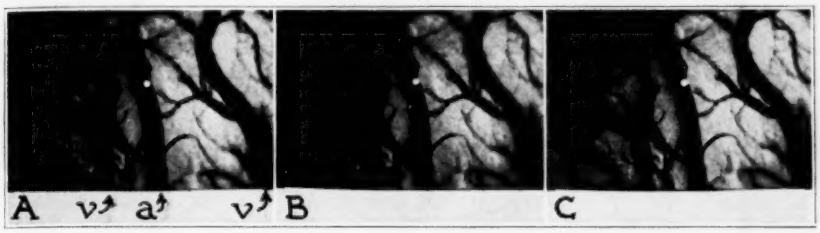

Fig. 2.—Photomicrographs (low power) of pial vessels in the isolated head taken with a Leica camera, Persenso film. Objective, × 3; ocular, × 10. *A* is the control; *B* was taken at the end of stimulation of the sympathetic nerve of thirty seconds' duration (coil distance, 12 cm.), and *C*, eighty seconds later; *a* indicates an artery; and *v* a vein.

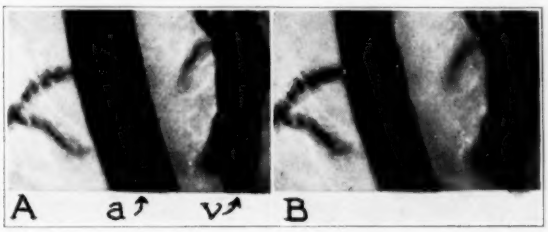

Fig. 3.—Photomicrographs (high power) made in the same experiment as those in figure 2. Objective, × 10; ocular, × 10. *A* is the control; *B* was taken during stimulation of the sympathetic nerve, forty-five seconds after stimulation started (coil distance, 12 cm.).

vessels. Stimulation of the cervical sympathetic nerve of the donor produced no perceptible change in the recipient's arteries.

2. Stimulation of the cervical sympathetic nerve in the recipient was invariably followed by dilatation of the ipsilateral pupil, the dilatation

6. Forbes, H. S.; Finley, K. H., and Nason, G. I.: Cerebral Circulation: XXIV. A. Action of Epinephrine on Pial Vessels; B. Action of Pituitary and Pitressin on Pial Vessels; C. Vasomotor Response in the Pia and in the Skin, Arch. Neurol. & Psychiat. **30**:957 (Nov.) 1933.

Effect of Stimulation of the Sympathetic Nerve in the Isolated Head

	Isolated Head								Donor			
	Stimulation of Sympathetic Nerve		Ipsilateral Pial Artery, Microns				Cerebrospinal Fluid Pressure		Blood Pressure, Mm. Hg			
Experiment	Coil Distance, Cm.	Duration, Sec.	Before Stimulation	After Stimulation	Per Cent Change	Ipsilateral Pupil	Before Stimulation	Per Cent Change	Before Stimulation	Per Cent Change	Anesthesia and Method	Comment
1	8	60	270.0	225.0	—17.0	..	..	....	106	+ 9	Dial, heparin; glass cannulas	Ringer's solution and epinephrine had been given because of low initial blood pressure (16 mm. Hg)
2	10	30	90.0	82.5	— 8.0	..	..	....	94	0	Dial, heparin; glass cannulas	Blood flow in observed artery somewhat retarded; many emboli present; further trials discarded because of poorer flow and more emboli
	10	55	97.5	90.0	— 8.0	..	..	....	94	0		
3	8	35	300.0	277.5	— 7.0	..	..	....	104	+29	Dial, heparin; glass cannulas	1st trial: flow slightly slowed; 2d trial: flow had momentarily stopped a few minutes before stimulation; 3d, 4th and 5th trials: good flow
	8	30	300.0	292.5	— 2.5	+†	..	....	120	0		
	8	30	292.5	270.0	— 8.0	+	..	....	114	0		
	9	30	292.5	258.75	—12.0	+	..	....	98	+22		
	12	30	285.0	270.0	— 5.0	+	..	....	74	+13		
4	11	30	131.25	112.5	—14.0	..	..	....	86	0	Dial, heparin; glass cannulas	Flow somewhat retarded, a few emboli present; further trials discarded because of many emboli and interrupted flow
	11	30	142.5	138.75	— 3.0	+	..	....	98	0		
5	11	45	105.0	97.5	— 7.0	+	..	....	80	+10	Dial, heparin; glass cannulas	Good flow in these 3 trials, flow then slower and many emboli present; no further response from stimulation of the sympathetic nerve
	11	30	135.0	112.5	—17.0	+	..	....	80	0		
	11	30	135.0	112.5	—17.0	+	..	....	74	0		
6	11 to 9	30 to 60	.....	.....	0	..	..	....	...	....	Amytal, heparin; glass cannulas	No perceptible change following stimulation of the sympathetic or vagus nerve; flow was intermittently slowed throughout

7	11	30	157.5	146.25	— 7.0	+	..	....	108	0	Dial; Crile's cannulas	Tracheal cannula in animal (B) found plugged with mucus; no further results with stimulation of the sympathetic nerve, probably because of insufficient oxygenation
8	12	30	120.0	120.0	0	+	..	....	66	0	Amytal; Crile's cannulas	Good flow throughout; artificial respiration; blood pressure abnormally low during first two trials
	11	60	120.0	120.0	0	+	..	....	66	0		
	11	60	225.0	206.25	— 8.0	..	..	....	74	+ 8		
9	12	25	292.5	262.5	—10.0	+	..	....	156	0	Dial; Crile's cannulas	Flow good throughout; graphic chart and photomicrographs appended
	12	30	292.5 (fig.2)	Photo	.....	+	..	....	150	0		
	12	45	322.5 (fig.3)	285.0	—12.0	+	..	....	106	0		
	12	30	270.0	240.0	—11.0	..	313	—14	88	0		
	12	30	262.5	228.75	—13.0	+	335	—13	90	0		
	12	30	273.75	240.0	—12.0	..	335	—15	104	0		
	12‡	30	270.0	240.0	—11.0	..	..	....	102	0	.....	After 0.1 mg. of ergotamine tartrate into vein of donor
	12‡	30	277.5	255.0	— 8.0	+	..	....	82	0	.....	After 2d dose of ergotamine tartrate (0.1 mg.) into vein of donor
10	12	60	135.0	120.0	—11.0	+	228	—12	100	+ 8	Amytal; Crile's cannulas	Flow good throughout
	12	30	135.0	127.5	— 6.0	+	228	—17	104	0		
	12	60	142.5	127.5	—11.0	+	240	—15	96	0		
	12‡	60	120.0	120.0	0	+	212	— 4	104	— 9	.....	After 0.05 mg. of ergotamine tartrate into left carotid of recipient
	11‡	60	120.0	116.25	— 3.0	+	205	— 3	92	—13		
	9‡	60	112.5	112.5	0	+	180	0	124	0	.....	After 2d dose of ergotamine tartrate (0.1 mg.)

† + = dilatation.

‡ Results from sympathetic stimulation after injection of ergotamine tartrate not included in averages.

usually occurring almost immediately after the onset of stimulation, whereas vasoconstriction of the pial vessels generally began after a further interval (from fifteen to thirty or more seconds). We interpreted this lag as being due to a more sensitive mechanism in the pupil than in the blood vessels. It was noticed also that the pupil responded to a much weaker stimulus than did the vessels.

3. Cerebrospinal fluid pressure in the isolated head exhibited an average fall (two experiments) of approximately 14 per cent, concomitant with stimulation of either the ipsilateral or the contralateral cervical sympathetic nerve, the effect presumably being due to decreased volume within the cranial vault occasioned by the diminished vascular bed. Approximately the same percentage of fall occurs in intact animals, the average for forty-six experiments being 12 per cent.[7]

4. On a few occasions (seven of twenty-six trials) the blood pressure of the donor rose slightly after stimulation of the sympathtic nerve in the recipient, amounting to an average rise of 13 mm. of mercury, or 15 per cent. This effect may have been due to a hormonal action or to temporarily increased resistance in the perfused head, dependent on the degree of vasoconstriction therein. It may have been merely accidental, for no dilatation of the donor's pupil was noticed.

5. Some allied observations follow:

A. Stimulation of the vagus nerve in the isolated head exerted no consistent effect on the ipsilateral pial arteries. In nine trials, vasodilatation resulted twice and vasoconstriction once, with no change six times. The recipient's spinal fluid pressure exhibited an increase four times, with no change once, while the blood pressure showed no change in eight of nine trials, with a slight decrease once. Stimulation of the donor's vagus nerve was followed by vasodilatation of the recipient's pial arteries four times and by no change once. The effect on the arterial pressure of the donor was a decrease on four occasions and no change once. This vasodilatation may have been due either to a chemical substance released by the vagal stimulation and carried over to the isolated head, or to an alteration in blood chemistry (retained carbon dioxide) consequent to changes in respiratory rate and depth. It is obvious that with so few data no definite conclusions can be drawn as to the effect of vagal stimulation in either animal.

B. The effect of epinephrine on the pial vessels of the isolated head was tested in four experiments (seven trials). In four of these trials the solution was injected into the carotid artery of the recipient. The pial artery immediately constricted. It then dilated when the

7. Forbes, H. S.: Unpublished data.

epinephrine reached the circulation of the donor, causing the blood pressure of the latter to rise. In three trials the solution was injected into the femoral vein of the donor. The pial artery of the isolated head showed a dilatation as the blood pressure rose; then a constriction, which increased as the blood pressure returned to normal. This corroborates previous work [8] which indicates that the direct action of epinephrine results in constriction of the pial arteries in the intact animal.

C. The effect of carbon dioxide was tested by producing temporary asphyxia in the donor through occlusion of the tracheal cannula (for one minute) and by direct inhalation of approximately 20 per cent carbon dioxide. The result in each instance was vasodilatation of the observed pial vessels, as might be expected from the work of Wolff and Lennox.[9]

CONCLUSIONS

Stimulation of the cervical sympathetic nerve in a completely isolated head, when directly and adequately perfused by the blood of a living intact animal, was followed by:

(*a*) Constriction of the ipsilateral arteries of the pia mater (average decrease in diameter 10 per cent).

(*b*) No change in diameter of the contralateral arteries of the pia.

(*c*) Dilatation of the epsilateral pupil.

(*d*) Decrease (averaging 14 per cent) in the cerebrospinal fluid pressure.

We believe that this experiment is crucial proving (in cats) the existence of a true vasoconstrictive action of the cervical sympathetic nerve on the ipsilateral pial arteries.

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8. Finesinger and Putnam.[4] Forbes, Finley and Nason.[6]

9. Wolff, H. G., and Lennox, W. G: Cerebral Circulation: XII. The Effect on Pial Vessels of Variations in the Oxygen and Carbon Dioxide Content of the Blood, Arch. Neurol. & Psychiat. **23**:1097 (June) 1930.

HERNIATION OF THE NUCLEUS PULPOSUS

A CAUSE OF COMPRESSION OF THE SPINAL CORD

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In 1927 and 1928 two patients with unusual cases of apparent extradural tumor of the spinal cord were operated on in University Hospital. The nodules which were removed were at first thought to be chondromas arising from intervertebral disks, but because microscopic examination showed no neoplastic tissue their nature and origin were for a time left in doubt. In the light of recent increase in knowledge concerning pathologic processes of the vertebral column, these nodules are now identified as herniations of nucleus pulposus tissue from intervertebral disks. Reports of these cases and a brief description of certain aspects of pathologic conditions of the disks follow.

REPORT OF CASES

CASE 1.—*Syndrome of involvement of cauda equina. Complete cerebrospinal fluid block. Localization with iodized oil. Laminectomy. Removal of nodule from intervertebral disk. Recovery.*

History.—F. R. H., salesman, aged 31, was referred by Dr. F. J. Graham in December 1926, complaining of pain in the back which radiated down both thighs. The onset had been one and a half years before, with pain and tingling in the calf of the right leg. Both legs eventually became weak and for three months previous to admission the patient was confined to bed. For two months there were difficulty in emptying the bladder, sexual impotence and some rectal incontinence. There was a loss of 48 (21.8 Kg.) pounds. No history of trauma was obtained.

Examination.—The heart, lungs and abdomen were normal. An enlarged prostate and hemorrhoids were noted. The lower part of the spine was held rigidly and there was loss of the normal lumbar curve. No palsies of the cranial nerves were found. The biceps and triceps reflexes were very active. There was obvious atrophy of the muscles of the lower extremities. The knee jerks were equal and active, and the achilles reflexes were absent. Plantar stimulation caused no movement of the toes on the right; on the left there was slight flexion. Sense of motion and position of the toes was absent on the right but retained on the left. Appreciation of pin point and light touch was impaired in the distribution of the fifth lumbar nerve and from the first to the fifth sacral nerves bilaterally.

Special Studies.—Routine examinations of the urine and blood gave negative results. A lumbar puncture between the third and fourth lumbar spinous processes

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yielded only 3 cc. of a quickly coagulating, xanthochromic fluid which contained 38 lymphocytes per cubic millimeter. The Queckenstedt test indicated a complete spinal block. The phenol reaction produced a heavy precipitate; the Wassermann test was negative, and the colloidal gold curve, 00112344220. Four days later, a puncture was made between the eleventh and twelfth thoracic vertebrae. As there was no evidence of spinal block at this level, descending iodized poppy-seed oil 40 per cent was injected. Lateral roentgenograms (fig. 1) showed a posterior narrowing of the intervertebral disk between the second and third lumbar vertebrae, and a rarefied area in the superior part of the third vertebral body (Schmorl

Fig. 1 (case 1).—In this roentgenogram, narrowing of the posterior part of the disk between the second and third lumbar vertebrae is clearly demonstrated. Most of the descending iodized oil has stopped at this level. The rarefied area in the body of the third vertebra represents invading nucleus pulposus tissue (Schmorl node).

node). As most of the iodized oil stopped at the level of this narrowed disk, a diagnosis was made of tumor pressing on the cauda equina at this point.

Operation.—Under ether anesthesia, the laminae and the posterior spinous processes of the first three lumbar vertebrae were removed. It was immediately evident that the cauda and the dura were displaced posteriorly at one point. When the dura was opened, the nerves of the cauda were found to be hyperemic and

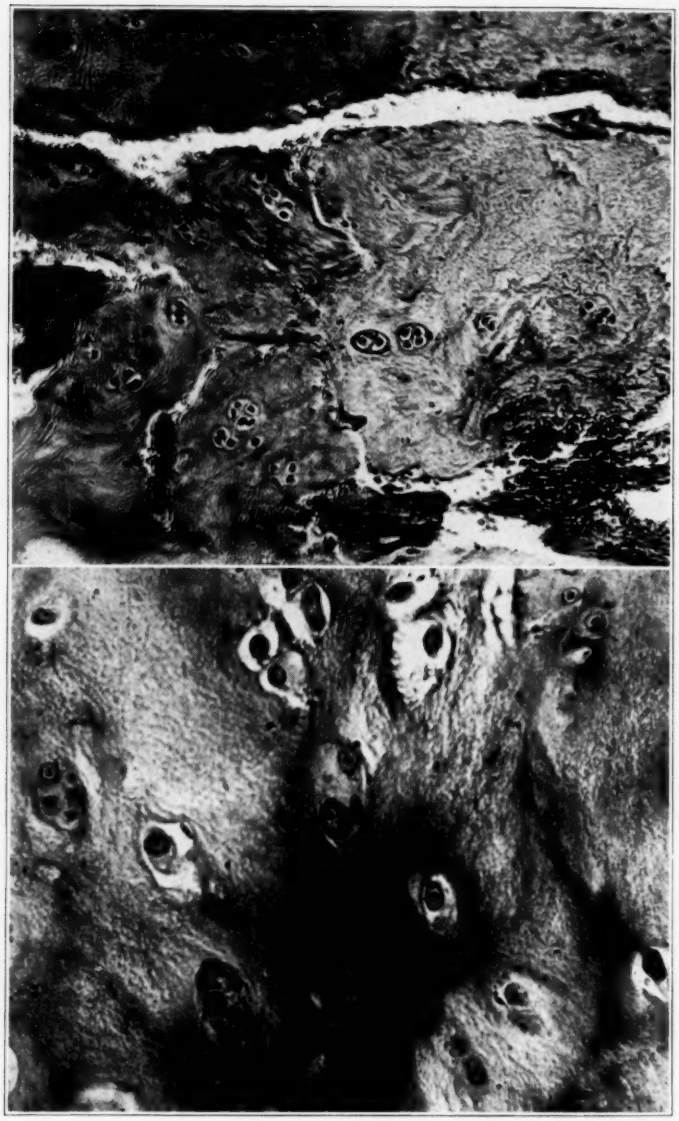

Fig. 2 (cases 1 and 2).—The upper and lower parts of the illustration show two nodules containing cartilage cells scattered through a relatively acellular matrix. They closely resemble the normal nucleus pulposus shown in figure 3. High power photomicrographs; hematoxylin and eosin stain.

glued together by a thickening of the arachnoid. These nerves were separated and a nodule was found anterior to the dura. The cauda and the dura were then retracted to one side and the nodule was exposed. It was 1 cm. in diameter and was attached to the disk between the second and third lumbar vertebrae. After the nodule was removed from its base by sharp dissection, a dural elevator was easily inserted into the substance of the disk. The dura was left open for decompressive purposes and the muscles were closed in layers.

Course.—At the time of discharge one month later, the patient was walking. He returned several times during the following year because of pain along the course of the left sciatic nerve. Some weakness and atrophy of the left leg persisted. Sexual power and urinary control were restored. When seen five and

Fig. 3.—Normal nucleus pulposus. High power magnification; hematoxylin and eosin stain.

three-fourth years after operation, the patient was working and in good health. He had no complaints except an occasional attack of sciatica.

CASE 2.—*Clinical signs of pressure on the cervical spinal cord. Partial subarachnoid block. Laminectomy and removal of a nodule from an intervertebral disk. Partial recovery.*

History.—E. H. H., a master mechanic, aged 49, was referred by Dr. J. D. Matthews in February 1928, complaining of soreness and weakness of the arms and difficulty in walking. The first symptom, slight swelling of both knees, had appeared one year before. This was followed by weakness of the legs. Later he noticed weakness of the right arm and then of the left hand, and finally soreness in all the extremities. There was no urinary difficulty.

Examination.—The patient was somewhat obese, with normal lungs, heart, abdomen and genitalia. The teeth were carious and the tonsils were hypertrophic and injected. The pupils were irregular in outline and unequal. No other abnor-

malities of the cranial nerves were present. There were weakness and spasticity of all four extremities. Hoffmann's sign was elicited on both sides. The biceps and triceps reflexes were exaggerated, and the abdominal reflexes were lost. The knee and ankle jerks were hyperactive, and there was a positive Babinski sign on both sides. Bilateral patellar and ankle clonus was elicited. Sense of motion and position of the toes was impaired on the right. Vibratory sense was lost in both ankles. Definite impairment of sensibility to light touch and pin prick was present below the level supplied by the fourth cervical segment.

Special Studies.—Routine studies of the urine and blood gave practically normal results. Lumbar puncture showed a partial subarachnoid block. The colorless fluid contained a normal number of cells and no increase in globulin. The Wassermann test was negative. The colloidal curve was 0011110000, and the

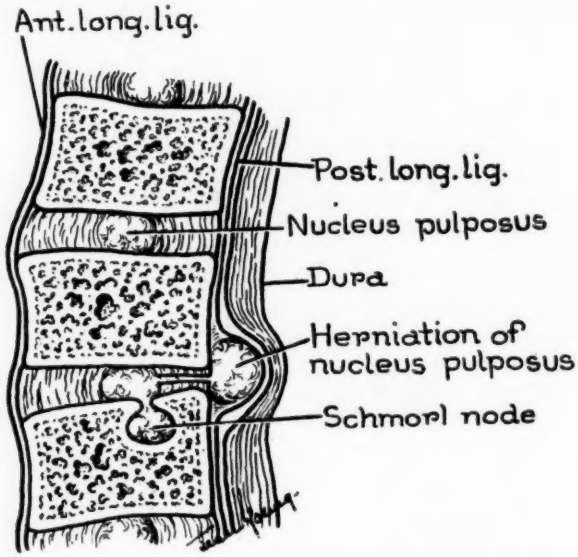

Fig. 4 (case 1).—Diagram showing herniation of nucleus pulposus into both the adjacent vertebral body and the spinal canal.

mastic curve was 111000. Lateral roentgenograms showed no abnormality of the vertebrae or of the intervertebral disks. The high pyramidal tract signs, the partial subarachnoid block and the sensory level at the fourth cervical segment suggested a midcervical tumor of the spinal cord.

Operation.—Under ether anesthesia the posterior spinous processes and laminae of the third, fourth, fifth and sixth cervical vertebrae were removed. The dura, which appeared to be normal, was incised in the midline. A posterior displacement of the cord at about the level of the fourth vertebra was immediately noticed. The vessels on this part of the cord were tortuous, enlarged and congested as compared with those higher up. On elevating the cord in this region, a yellowish-white nodule was seen anterior to the dura. On opening the dura, a smooth, firm, encapsulated mass was disclosed. It was bean-shaped, was attached to the intervertebral disk between the third and the fourth vertebra, and extended from the midline to the right, pressing on the nerve root at that

level. On incising the capsule, cartilaginous material was spontaneously extruded. After curetting out the remainder of this tissue the cord was replaced and the posterior layer of the dura was closed with a continuous suture. The muscles were closed in layers.

Course.—A few days after the operation the patient began to regain strength in the arms, and sensation rapidly improved. Following removal of the sutures on the seventh day, the wound reopened and a secondary closure was done. The patient was discharged at the end of one month. When last heard from, four and one-half years after the operation, he was in good health and was working as a carpenter. He was completely free from pain and had regained the use of his legs but carried a cane because of slight unsteadiness.

COMMENT

At its circumference, each intervertebral disk is composed of laminae of dense fibrocartilage forming the annulus fibrosus. The fibers run obliquely from one vertebra to the next and are firmly attached to them. The nucleus pulposus, an incompressible, semigelatinous mass, is found near the center of the disk. Interposed between the disk and the adjacent vertebrae are thin plates of hyaline cartilage. Histologically, the nucleus is composed of loose fibrous tissue, a few cartilage cells, some remnants of the notochord (large multinuclear cells) and a gelatinous matrix. The nucleus pulposus, confined to its position by the elastic annulus fibrosus and the two cartilage plates, acts as a shock absorber and a hydrodynamic ball bearing for the spine.

Little was known of the pathology of the intervertebral fibrocartilage until 1925, when Schmorl [1] began to remove every spine for examination as a routine of autopsy. As a result of his work and that of others,[2] considerable knowledge has accumulated regarding the escape of the nucleus pulposus from the intervertebral disk in abnormal conditions. For example, local degeneration or trauma may produce a minute fissure in the annulus fibrosus or in a cartilage plate. In each

1. Schmorl, G.: Die pathologische Anatomie der Wirbelsäule, Verhandl. d. deutsch. orthop. Gesellsch. **21**:3, 1926; Ueber die an den Wirbelbandscheiben vorkommenden Ausdehnungs- und Zerreissungsvorgänge und die dadurch an ihnen und der Wirbelspongiosa hervorgerufenen Veränderungen, Verhandl. d. deutsch. path. Gesellsch. **22**:250, 1927; Ueber Knorpelknoten an der Hinterfläche der Wirbelbandscheiben, Fortschr. a. d. Geb. d. Röntgenstrahlen **40**:629, 1929.

2. (*a*) Andrae, R.: Ueber Knorpelknötchen an hinteren Ende der Wirbelbandscheiben im Bereich des Spinalkanals, Beitr. z. path. Anat. u. z. allg. Path. **82**:464, 1929. (*b*). Beadle, O. A.: The Intervertebral Discs: Observations on Their Normal and Morbid Anatomy in Relation to Certain Spinal Deformities, Medical Research Council, Spec. Rep. Ser. 161, London, His Majesty's Stationery Office, 1931. (*c*). Calvé, J., and Galland, M.: The Intervertebral Nucleus Pulposus: Its Anatomy, Its Pathology, Its Physiology, J. Bone & Joint Surg. **12**:555, 1930. (*d*). Sashin, D.: Intervertebral Disk Extensions Into the Vertebral Bodies and the Spinal Canal, Arch. Surg. **22**:527 (April) 1931.

instance, the nucleus pulposus, which is always under pressure, herniates through the defect. In the case of the fissured cartilage plate, the nuclear material forces its way into the cancellous bone of the vertebral body. This invading substance then undergoes proliferation and organization and finally results in a firm nodule of cartilage. Also, a wall of dense bone is laid down about the lesion, making it visible in roentgenograms (fig. 1). Keyes and Compere [3] reproduced this condition in dogs and monkeys by passing a drill through the vertebral body into the intervertebral disk. The fissures which develop in the annulus fibrosus are usually situated posteriorly. Consequently, the prolapsing nuclear substance enters the spinal canal, where it forms a swelling under the posterior longitudinal ligament, usually to one side of the midline. Here, also, secondary changes take place, transforming the herniated material into a firm nodule of cartilage which usually remains attached to the disk. This condition is fairly common. Andrae,[2a] in a series of 368 autopsies on the spine, found 56 cases of posterior nuclear extrusions which ranged in size from that of a hemp seed to that of a bean. All but 1 of these were too small to have produced compression of the spinal cord. In 16 of these cases, selected at random, he was able,[4] by serial sectioning, to demonstrate the connection between the nodule of cartilage and the center of the disk.

Grossly, the nodules in our cases resembled those described by Andrae except that his were smaller. Histologically, our nodules resembled normal nucleus pulposus (figs. 2 and 3). They showed a few cartilage cells scattered through a relatively acellular matrix. There was no inflammatory change or suggestion of neoplasm. Therefore, we have no hesitation in identifying them as herniations of nucleus pulposus.

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3. Keyes, D. C., and Compere, E. L.: The Normal and Pathologic Physiology of the Nucleus Pulposus of the Intervertebral Disk, J. Bone & Joint Surg. **14**:897, 1932.

4. Through an unfortunate error in translation, this work of Andrae was misquoted by Alpers, Grant and Yaskin.[9] In discussing the possibility of prolapse of the nucleus pulposus they stated: "It seems hardly likely that this is the case, because in sixteen cases which Andrae found there were no direct connections between the disk extension and the nucleus pulposus. There was always intact annulus fibrosus between them." Andrae actually said: "In sixteen of those intervertebral disks which I have cut in serial sections, it was regularly possible to establish a connection of the cartilage nodules with the central part of the disk, i. e., the nucleus pulposus. In almost all cases the nucleus pulposus and the posterior parts of the annulus fibrosus were destroyed or degenerated." The exact statement is as follows: "Bei sechzehn von diesen Bandscheiben, die ich in Serien schnitt, gelang es mir regelmässig, einen Zuzammenhang der KK. (Knorpelknötchen) mit den zentralen Bandscheibenpartien bzw. den Nucl. pulp. nachzuweisen. Bei weitaus der Mehrzahl der Fälle waren die Bandscheiben selbst—d.h. ihr Nucl. pulp. und die hinteren Abschnitte des Annulus fibrosus—in Zerstörung oder Degeneration begriffen. . . ."

In our first case, lateral roentgenograms (fig. 1) showed a narrowing of the disk from which the nucleus herniated. In addition, the adjacent vertebral body showed a rarefied area which indicated that some nuclear substance had also ruptured into it (fig. 4). It may be stated that if a clinical diagnosis of compression of the spinal cord has been established and if there is evidence of a diseased intervertebral disk at the proper level, retropulsion of the nucleus pulposus should be considered.

About 50 cases of nodules surgically removed from disks have been reported, one half of them having been diagnosed chondroma or fibrochondroma. Since nodules of the posterior part of the disks of nucleus pulposus origin are a firmly established pathologic entity, we think that neurosurgeons should cease to describe them under such misleading terms. They represent hernias and not new growths. Elsberg,[5] who found 14 nodules of the disk in a series of 100 tumors of the spinal cord, showed that histologically his material corresponded with normal nucleus pulposus. However, although he specified that they were cartilage hyperplasias and not neoplasms, he continued to use the term chondroma. In Dandy's [6] 2 cases and in some of Elsberg's cases the cartilage bodies were not attached to the disks but could be lifted out as soon as the overlying dura and ligamentous fibers were incised. This is strong evidence that the lesions represented extrusions from the disks (as Dandy explained) and not tumors. Stookey [7] and Adson [8] reported numerous cases as chondromas, but this was before the rôle of the nucleus was known. Alpers, Grant and Yaskin [9] (1 case) and Bucy [10] (1 case) also used the term chondroma, although areas of their specimens resembled nucleus pulposus. Probably the first to favor rupture of the nucleus pulposus were Alajouanine and Petit-Dutaillis,[11] who reported 2 cases in 1930. More recently, at a meeting of the Boston Orthopedic Club (Dec. 11, 1933), Barr reported 18 operative cases from the Massachusetts General Hospital files and identified the lesions histologically as prolapsed disk substance.

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5. Elsberg, C.: The Extradural Ventral Chondromas, Bull. Neurol. Inst. New York **1**:350, 1931.

6. Dandy, W. E.: Loose Cartilage From the Intervertebral Disk Simulating Tumor of the Spinal Cord, Arch. Surg. **19**:660 (Oct.) 1929.

7. Stookey, B.: Compression of the Spinal Cord Due to Ventral Extradural Cervical Chondromas, Arch. Neurol. & Psychiat. **20**:275 (Aug.) 1928.

8. Adson, A. W.: Diagnosis and Treatment of Tumors of the Spinal Cord, Northwest Med. **24**:309, 1925.

9. Alpers, B. J., Grant, F. C., and Yaskin, J. C. Chondroma of the Intervertebral Disk, Ann. Surg. **97**:10, 1933.

10. Bucy, P. C.: Chondroma of Intervertebral Disk, J. A. M. A. **94**:1552 (May 17) 1930.

11. Alajouanine, T. and Petit-Dutaillis, D.: Le nodule fibro-cartilagineux de la face postérieure des disques inter-vertebraux, Presse méd. **98**:1657, 1930.

SUMMARY

1. Two patients with nodules on intervertebral disks producing symptoms of tumor of the spinal cord recovered following surgical excision.

2. The nodules removed were not tumors but consisted of nucleus pulposus tissue which had undergone secondary changes following herniation from the intervertebral disks.

3. Herniated nucleus pulposus should be considered in cases of compression of the spinal cord which present roentgen evidence of a diseased disk at the proper level.

THE NEUROFIBRILS IN SYSTEMIC DISEASE AND IN SUPRAVITAL EXPERIMENTS

WITH REMARKS ON PSEUDO-ATROPHY OF THE BRAIN

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Studies of cerebral changes in dysentery and other severe gastrointestinal infections, carried out in collaboration with T. T. Wu, made it necessary, before judging the results, to study some features in specimens stained by the Bielschowsky method which hitherto have been considered pathologic, such as thickening, formation of more or less winding strands and especially argentophilia of intracellular neurofibrils in supravital experiments. Studies of the behavior of the brain tissue by supravital incubation in different fluids are not new. Zanke [1] used this method to determine the liability of the brain tissue to swell in fluids of different tonicity; he carried on his studies by comparative weights. He found an increase in swelling of human brain tissue of from 15 to 20 per cent in water and of from 7 to 10 per cent in 0.6 per cent saline solution. Apelt [2] studied whether this swelling would be different under different pathologic conditions. He came to the conclusion that after incubation in 0.9 per cent saline solution for twenty-four hours edematous brains gain as much weight as normal nonedematous brains. According to Apelt this indicates that in these cases the swelling of the brain is not simple edema. It might also be of importance that, according to Reichardt, the normal brain is already very rich in water (70 per cent for the white, and 83 per cent for the gray matter). Therefore a change in the water content may not be gross enough to cause a difference in the swelling sufficiently great to be measurable with the scale and to be beyond the limits of error.

Histologic studies of the results of experimental postmortem swelling of the brain have been performed by Weed and McKibben [3] and

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1. Zanke: Ueber Messung des Schädelinnenraumes, Neurol. Centralbl. **16:** 488, 1897; quoted by Apelt; [50] Hirngewicht und Schädelinnenraum, Neurol. Centralbl. **16:**881, 1897; quoted by Apelt.[50]

2. Apelt, F.: Weitere mikroskopische und physikalische Untersuchungen der Hirnsubstanz zur Frage nach der Ursache der Hirnschwellung, Deutsche Ztschr. f. Nervenh. **39:**119, 1910.

3. Weed, L. H., and McKibben, P. S.: Experimental Alteration of Brain Bulk, Am. J. Physiol. **48:**531, 1919.

by Fortuyn.[4] Their most important result is the observation that the chief postmortem process of swelling takes place in the intercellular substance, while the cells themselves even shrink. The cytologic studies were performed only with nuclear and protoplasmic stains; Fortuyn,[5] especially, could make out two different types, inflated and noninflated cells, but no silver preparations were examined. Rose,[6] in his instructive studies on the influence of fixation on the cellular picture of the cerebral cortex, confined himself to the Nissl picture.

The question of neurofibrillar changes, however, is one of the most unsettled and undecided in modern neuropathologic literature. Therefore, there appeared to exist a need for an experimental study which would determine the influence of the postmortem application of fluids of different tonicity on the impregnation of neurofibrils.

Neurofibrillar changes are described in the most different kinds of pathologic and even physiologic conditions. Thickening, argentophilia and formation of more or less winding strands were found to be an important feature of senile and presenile changes in the brain in the classic studies of Bielschowsky and Brodmann,[7] Alzheimer [8] (who especially emphasized the formation of winding argentophil strands, which were later called after him the "Alzheimer change of neurofibrils"), Bielschowsky,[9] Perusini,[10] Simchowicz,[11] Montesano [12] and Fischer.[13]

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4. Fortuyn, A. B. D.: Histological Experiments with the Brain of Some Rodents, J. Comp. Neurol. **42**:349, 1927.

5. Fortuyn, A. B. D.: Changements histologiques dans l'écorce cérébrale de quelques rongeurs, Trav. du lab. de recherches biol. de l'Univ. de Madrid **22**:67, 1924; footnote 4.

6. Rose, M.: Ueber den Einfluss der Fixierung auf des Zellbild der Grosshirnrinde, J. f. Psychol. u. Neurol. **38**:155 (Feb.) 1929.

7. Bielschowsky, M., and Brodmann, K.: Zur feineren Histologie und Histopathologie der Grosshirnrinde mit besonderer Berücksichtigung der progressiven Paralyse, J. f. Psychol. u. Neurol. **5**:173, 1905.

8. Alzheimer, A.: (*a*) Ueber eine eigenartige Erkrankung der Hirnrinde, Zentralbl. f. Nervenh. u. Psychiat. **30**:449, 1907; (*b*) Ueber eigenartige Krankheitsfälle des späteren Alters, Ztschr. f. d. ges. Neurol. u. Psychiat. **4**:356, 1911.

9. Bielschowsky, M.: (*a*) Zur Kenntnis der Alzheimerschen Krankheit, J. f. Psychol. u. Neurol. **18**:273, 1911; (b) Beiträge zur Histopathologie der Ganglienzellen, J. f. Psychol. u. Neurol. **18**:513, 1912.

10. Perusini, G.: Ueber klinisch und histologisch eigenartige Erkrankungen des späteren Lebensalters, Histol. u. histopath. Arb. ü. d. Grosshirnrinde **3**:297, 1909.

11. Simchowicz, T.: Histologische Studien über die senile Demenz, Histol. u. histopath. Arb. ü. d. Grosshirnrinde **4**:271, 1911.

12. Montesano, G.: Ueber die von Alzheimer beschriebene Neurofibrillenveränderung in Form von argentophilen Kügelchen, Ztschr. f. d. ges. Neurol. u. Psychiat. **3**:720, 1911.

13. Fischer, O.: Ein weiterer Beitrag zur Klinik und Pathologie der presbyophrenen Demenz, Ztschr. f. d. ges. Neurol. u. Psychiat. **12**:99, 1912.

Recently, Fluegel [14] found these changes also in the spinal cords of senile persons. But neurofibrillar changes have also been described in other conditions. In dementia paralytica Bielschowsky and Brodmann,[7] Raecke [15] and Alzheimer [16] found fibrillar changes in the form of thickening, argentophilia and breaking into short plump little rods or small granules. Hallervorden [17] found even the true and fully developed Alzheimer change of neurofibrils in chronic cases of epidemic encephalitis. Lewy [18] observed the same change in paralysis agitans. Achúcarro [19] found thickening and argentophilia of neurofibrils in rabies in animals and in man. Barrett [20] and Schaffer [21] described Alzheimer changes of neurofibrils in the brains in cases of spastic spinal paralysis in patients 33 and 28 years of age. A similar case is also mentioned by von Braunmühl.[22] Van Bogaert and Bertrand [23] described Alzheimer changes in two cases of amyotrophic lateral sclerosis occurring at 46 and 59 years of age. Lafora [24] described Alzheimer changes of intracellular neurofibrils in the cornu ammonis of a Negro woman aged 46, who had suffered for ten years from a characteristic schizophrenic psychosis. Finally, parenchymatous nephritis and heart failure developed, with a gradually increasing general anasarca. Autopsy revealed marked edema of the brain. Lafora considered the possibility of an influence of anasarca in the etiology of the histologic changes of senile type. Following other diseases the

14. Fluegel, F. E.: Quelques recherches anatomiques sur la dégénérescence sénile de la moelle épinière, Rev. neurol. **1**:618 (May) 1927.

15. Raecke: Das Verhalten der Neurofibrillen bei der progressiven Paralyse, München. med. Wchnschr. **53**:1685, 1906.

16. Alzheimer, A.: Ergebnisse auf dem Gebiete der pathologischen Histologie der Geistesstörungen, Ztschr. f. d. ges. Neurol. u. Psychiat. (Ref.) **5**:753, 1912.

17. Hallervorden, J.: Zur Pathogenese des postencephalitischen Parkinsonismus, Klin. Wchnschr. **12**:692, 1933.

18. Lewy, F. H.: Die Lehre von Tonus und der Bewegung, in Alzheimer, A., and Lewandowsky, M.: Monographien aus dem Gesamtgebiete der Neurologie und Psychiatrie, Berlin, Julius Vogel, 1923, no. 34.

19. Achúcarro: Zur Kenntnis der pathologischen Histologie des Zentralnervensystem bei Tollwut, Histol. u. histopath. Arb. ü. d. Grosshirnrinde **3**:143, 1909.

20. Barrett, A. M.: A Case of Alzheimer's Disease with Unusual Neurological Disturbances, J. Nerv. & Ment. Dis. **40**:361, 1913.

21. Schaffer, K.: Zur Pathologie und pathologischen Histologie der spastischen Heredodegeneration (hereditäre spastische Spinalparalyse), Deutsche Ztschr. f. Nervenh. **73**:101, 1922.

22. von Braunmühl, A.: Kolloidchemische Betrachtungsweise seniler und praeseniler Gewebsveränderungen. Das hysteretische Syndrom als cerebrale Reaktionsform, Ztschr. f. d. ges. Neurol. u. Psychiat. **142**:44, 1932.

23. Van Bogaert, L., and Bertrand, I.: Pathologic Changes of Senile Type in Charcot's Disease, Arch. Neurol. & Psychiat. **16**:263 (Sept.) 1926.

24. Lafora, G. R.: Zur Frage des normalen und pathologischen Senium und der Pathologie der Senilität, Ztschr. f. d. ges. Neurol. u. Psychiat. **13**:469, 1913.

special metabolic changes of senility may develop much earlier than in normal persons. Malamud and Lowenberg [25] described, under the name of Alzheimer change, the occurrence of thickening, clubbing and fragmentation of the intracellular neurofibrils of the cortex in the case of a man, aged 23, who as a boy, after an attack of scarlet fever at the age of 7, had become mentally changed and demented; he died of pulmonary tuberculosis in a state hospital.

Neurofibrillar changes are recorded not only in different cases of definite diseases of the brain, but in various systemic diseases as well. Chanutina [26] described breaking up of the intracellular neurofibrils of the spinal cord into small pieces and granules in cases of Asiatic cholera. Michailow [27] described neurofibrillar changes throughout the whole brain cortex, brain stem and spinal cord in cholera asiatica, such as thickening, argentophilia and adhering together of neurofibrils. The thickened neurofibrils showed also slight winding or they formed strands, which ran along the margin or across through the cell. Besides these forms, cells were also observed in which the neurofibrils were broken or entirely destroyed. Parhon and Papinian [28] found fragmentation of the intracellular neurofibrils in the brain cortex and spinal cord in pellagra; Valtorta [29] also described fibrillar changes of different degrees in this disease. Rezza [30] described the formation of interlaced strands, especially along the margins of the cells, as well as crumbling of intracellular neurofibrils in pellagra psychoses, but he emphasized that no characteristic Alzheimer change could be observed. Kleist [31] found crumbling of the intracellular neurofibrils in a woman, aged 48, who had suffered from a symptomatic psychosis in pseudouremia. Hechst [32] described disruption of the intracellular neurofibrils into argentophil granules in one case of uremia and simple destruction

25. Malamud, W., and Lowenberg, K.: Alzheimer's Disease: A Contribution to Its Etiology and Classification, Arch. Neurol. & Psychiat. **21**:805 (April) 1929.

26. Chanutina: Zur Frage über die Veränderungen des Rückenmarks bei der Cholera, Russk. vrach. **34** and **35**: 1909.

27. Michailow, S.: Pathologisch-anatomische Untersuchungen der feineren Struktur der Gehirnrinde, der Rinde des Kleinhirns, des verlängerten und des Rückenmarks des Menschen bei asiatischer Cholera, Arch. f. Psychiat. **51**:587, 1913.

28. Parhon, R., and Papinian, J.: Note sur les altérations des neurofibrilles dans la pellagre, Compt. rend. Soc. de biol. **58**:360, 1905.

29. Valtorta, D.: Sulle alterazioni delle cellule nervose corticali in un caso di tifo pellagroso, Riv. pellagrol. ital. **8**:58 and 74, 1908.

30. Rezza, A.: Beitrag zur pathologischen Anatomie der Pellagrapsychosen, Ztschr. f. d. ges. Neurol. u. Psychiat. **12**:1, 1912.

31. Kleist, K.: Psychosen bei Stoffwechselstörungen, in Herxheimer, G.: Stoffwechselkrankheiten, Berlin, S. Karger, 1926.

32. Hechst, B.: Ueber Gehirnbefunde bei urämischen Zuständen, Ztshr. f. d. ges. Neurol. u. Psychiat. **139**:544, 1932.

in another. Donaggio [33] found a fibrillar change resembling the Alzheimer change in the brains of animals which had died of hunger and exposure to cold. Tello [34] found thickening and argentophilia of the intracellular neurofibrils even following the physiologic hibernation of reptiles.

These facts gave rise to the contention that the senile and presenile neurofibrillar changes should also be considered as metabolic in origin,[35] an opinion that found its most extensive theoretical motivation in the study of von Braunmühl.[22] He based his conclusions especially on the experiments of Bozler, who could change the neurofibrils in Rhizostomata into winding strands by incubation of the animals in hypertonic sea water. Von Braunmühl repeated these experiments with rubber models and benzene, but did not succeed in repeating them with human brains. He explained Bozler's phenomenon, who himself explained it otherwise, as due to swelling and compared it with the Alzheimer change of neurofibrils. Von Braunmühl explained the Alzheimer change as due to hydration and swelling of structures previously dehydrated in life. The cause of this change in the colloidal medium, leading to hysteresis and syneresis of the protoplasm, of which the senile neurofibrillar changes are one special form, may have metabolic as well as endocrine sources. He pointed especially to the results of Ruzicka,[36] who could produce increase of protoplasmic hysteresis of the skin in starving tritons similar to that of old age. Von Braunmühl went so far as to consider that the discovery of the metabolic or endocrine source of the Alzheimer change of neurofibrils might be of therapeutic importance, because it would offer measures to rejuvenate the brain.

Data concerning the relationship of endocrine disorders to neurofibrillar changes in the brain are already contained in the literature.

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33. Donaggio, A.: Effetti dell azione combinata del digiuno e del freddo sui centri nervosi di mammiferi adulti, Riv. sper. di freniat. **32**:373, 1906.

34. Tello, F.: Los neurofibrilles en los vertebrados inferiores, Trab. d. lab. de invest. biol. Univ. de Madrid **3**: 1904.

35. Amaurotic idiocy has similarly been determined to be only a part of a systemic disease of the organism by Bielschowsky (Amaurotische Idiotie und lipoidzellige Splenomegalie, J. f. Psychol. u. Neurol. **36**:103, 1928) and Kufs (Ueber die Konstitutions- und Vererbungspathologischen Grundlagen der Kombination der lipoidzelligen Splenohepatomegalie [Niemann-Pick] mit der infantilen Form der amaurotischen Idiotie, Ztschr. f. d. ges. Neurol. u. Psychiat. **117**:753, 1928).

36. Ruzicka, V.: Beschleunigung der Häutung durch Hunger, Arch. f. Entwicklgsmechn. d. Organ. **42**:671, 1917; Beiträge zum Studium der Protoplasmahysteresis und der hysteretischen Vorgänge. (Zur Kausalität des Alterns), Arch. f. mikr. Anat. **101**:459, 1924.

Schnitzler [37] found Alzheimer change of intracellular neurofibrils, argentophil balls, but no druses in a woman, 36 years of age, who at 31 years of age had experienced a gradually progressing mental change with dementia, together with myxedematous changes in the skin. Schob and Güntz [38] reported a case of pituitary cachexia with dementia in a woman, aged 64, which showed Alzheimer change of intracellular neurofibrils, as a combination of Alzheimer's and Simmonds' diseases. In experiments with animals Balli [39] could produce thickening, clumping and argentophilia of the intracellular neurofibrils in dogs by complete extirpation of the thyroid and parathyroid glands and subsequent exposure to cold of —7 C. for seven days. F. H. Lewy [18] confirmed Balli's observation by similar experiments with dogs, rabbits and apes. He could produce the fibrillar changes also by combined extirpation of the thyroid and parathyroid without subsequent freezing, or merely by extirpation of either the thyroid or the parathyroid with additional freezing at — 5 C. Concerning the pathogenesis of the fibrillar change, he thought that the disorder of the calcium metabolism, caused by deficiency of the parathyroid, leads to the formation of deposits of argentophil substances in the tissue and on the intracellular neurofibrils, thickened and adherent together owing to the loss of the thyroid. He drew an analogy between the neurofibrillar change produced in his experiments and the senile or presenile Alzheimer change and emphasized the importance of the thyroid and other endocrine glands for the etiology of the senium. In similar experiments Rasdolsky [40] found the same neurofibrillar changes as Balli and Lewy, but only in thyroidectomized animals which survived the extirpation for a certain length of time. He emphasized the statement that in these cases the degree of the changes of intracellular neurofibrils is proportional to the development and degree of cachexia. In the thyroparathyroidectomized animals which died within the first days after operation the silver stain showed only indistinct and undifferentiated blackish-brown masses in the cells.

Impressed by such facts, Grünthal [41] concluded that the neurofibrillar

37. Schnitzler, J. G.: Zur Abgrenzung der sogenannten Alzheimerschen Krankheit, Ztschr. f. d. ges. Neurol. u. Psychiat. **7**:34, 1911.

38. Schob and Güntz: Alzheimersche Krankheit kombiniert mit Simmondscher Erkrankung (hypophysärer Kachexie), Allg. Ztschr. f. Psychiat. **97**:242, 1932.

39. Balli, R.: Lesioni del reticulo neurofibrillare endocellulare in mammiferi adulti totalmente o parzialmente privati dell apparecchio tiro-paratireoideo e loro rapporto colla temperatura, Riv. Sper. di freniat. Arch. ital. per le mal. nerv. e ment. **32**:803, 1906.

40. Rasdolsky, J.: Histologische Veränderungen in dem zentralen und peripherischen Nervensystem der Tiere mit exstirpierten Schild- und Nebenschilddrüsen, Ztschr. f. d. ges. Neurol. u. Psychiat. **106**:96, 1926.

41. Grünthal, E.: Ueber die Alzheimersche Krankheit. Eine histopathologisch-klinische Studie, Ztschr. f. d. ges. Neurol. u. Psychiat. **101**:128, 1926.

changes in senile and presenile diseases are caused only indirectly by old age, and Hilpert [42] emphasized toxic influences on the basis of pathologic endocrine changes as factors in their development. This seems to be in accordance with Metchnikoff's [42a] views that the senium is due to poisoning by toxic products developed increasingly by the intestinal tract during human life and damaging the muscles, glands and the central nervous system. And, as concerns the relationship between the senium and endocrine disorders, Wegelin [43] showed that after acquired athyreosis changes which must be considered as premature involutions take place in the skin, arteries, bone marrow and genitals.

The experimental observations of Balli, F. H. Lewy and Rasdolsky, however, were not confirmed with human material in the careful studies of Lotmar [44] on the brain in endemic cretinism, congenital myxedema (thyroaplasia) and cachexia thyropriva. He found only slight argentophilia of the intracellular neurofibrils, but no thickening; only at rare places did the neurofibrils adhere together. No Alzheimer change or similar condition was found. Therefore Lotmar [44b] rejected the contention that there is a direct relationship between hypothyreosis or athyreosis and neurofibrillar changes. The assumption of some authors that the normal, the premature or the pathologic senium may be caused by hypothyreosis does not receive any support from the histologic observations on the brain in cases of congenital or acquired athyreosis or in cretinism.

The statements of Lotmar led us to suppose that all these different occurrences of neurofibrillar change are caused by a common, but unknown, factor which interferes with the result of the neurofibrillar impregnation.

EXPERIMENTS

The brain of a man, aged 26, who died of peritonitis following appendectomy on Aug. 26, 1933, was removed from the skull forty and three-fourths hours after death. Pieces of adjacent regions were cut out, and each one was incubated in one of the following fluids for seventeen hours: (1) solution of formaldehyde, U.S.P. (1:10), (2) distilled water, (3) 0.3 per cent saline solution, (4) 0.9 per cent saline solution, (5) 20 per cent saline solution, (6) solution of formaldehyde, U.S.P. (1:10), acidulated with acetic acid (20 drops of concentrated glacial acetic

42. Hilpert, P.: Zur Klinik und Histopathologie der Alzheimerschen Krankheit, Arch. f. Psychiat. **76**:379, 1926.

42a. Metchnikoff, E.: Etude sur la nature humaine, essai de philosophie optimiste, Paris, Masson & Cie, 1903.

43. Wegelin, C.: Schilddrüse, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie, Berlin, Julius Springer, 1926, vol. 8, pt. 1.

44. Lotmar, F.: (*a*) Histopathologische Befunde in Gehirnen von kongenitalem Myxödem. (Thyreoplasie), Ztschr. f. d. ges. Neurol. u. Psychiat. **119**:491, 1929; (*b*) Histopathologische Befunde in Gehirnen von endemischen Kretinismus, Thyreoaplasie und Kachexia thyreopriva, ibid. **146**:1, 1933.

acid in 65 cc. of solution of formaldehyde, U.S.P. [1:10]) and (7) solution of formaldehyde, U. S. P. (1:10), made alkaline with sodium hydroxide (20 drops of 40 per cent solution of sodium hydroxide in 65 cc. of solution of formaldehyde, U.S.P. [1:10]).

At first the macroscopic changes of the different pieces were noted; unfortunately the pieces were not weighed. The pieces in solution of formaldehyde, U.S.P. (1:10), did not show any deformity, even after the longest period of time. The pieces in distilled water and those in 0.3 and in 0.9 per cent saline

Fig. 1.—Section from the brain of a male patient, aged 26, who died of peritonitis. The specimen (frontal cortex, inferior part of the third layer) was fixed in solution of formaldehyde, U.S.P. (1:10), immediately after removal from the skull. Silver impregnation according to Bielschowsky; normal Bielschowsky picture; reduced from × 445.

solution showed swelling after about five hours. The pieces in alkaline solution of formaldehyde showed marked swelling and yellowish decoloration, even after half an hour. Those in acid solution of formaldehyde were markedly shrunken, even after about half an hour. The pieces in 20 per cent saline solution were moderately shrunken after about five hours.

After seventeen hours of incubation in the different fluids, all the pieces, with exception of the first, were fixed in solution of formaldehyde, U.S.P. (1:10) for fourteen days. Then frozen sections were prepared, and Bielschowsky's silver impregnation of intracellular neurofibrils was carried out according to the usual method and the directions laid down in the last edition of Spielmeyer's [45] book on histologic technic.

The pieces from adjacent regions of the brain cortex, incubated before fixation in the different fluids, were always stained at the same time and remained exactly the same length of time in the different reagents, which were prepared at the same time in big jars and then divided into seven equal portions. Thus, except for the supravital incubation, exactly the same technic was applied to all the specimens.

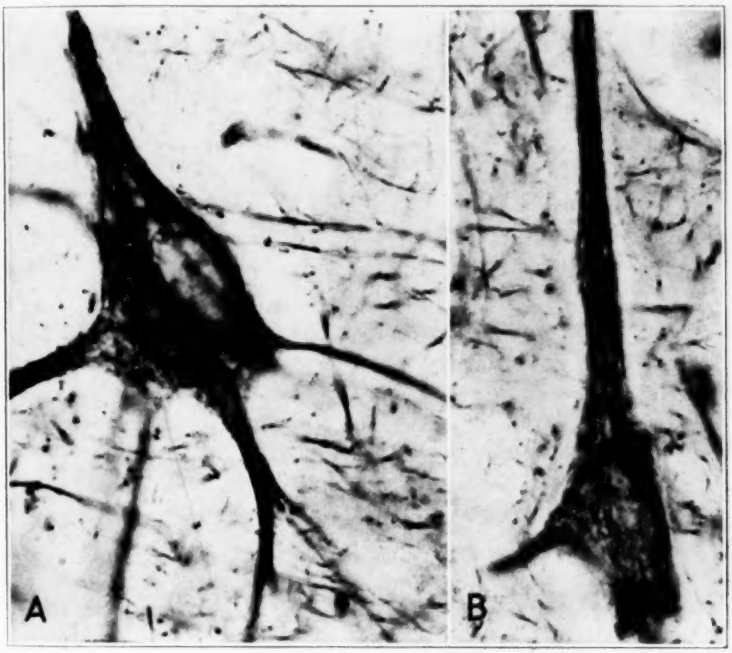

Fig. 2.—Two ganglion cells from the section shown in figure 1. Normal neurofibrillar structure and impregnation; nuclei light and unimpregnated; specimen fixed in solution of formaldehyde, U.S.P. (1:10); silver impregnation according to Bielschowsky; × 1,250, oil immersion.

The specimens removed from the different fluids showed marked differences microscopically. These differences must be considered with certainty as caused by the supravital incubation in the different fluids.

Without exception all the specimens fixed originally in solution of formaldehyde, U.S.P. (1:10), gave an ideally normal Bielschowsky stain (figs. 1 and 2). The intracellular neurofibrils and the axis-cylinders were nicely impregnated to a normal degree, with the characteristic blackish brown shade of good specimens.

45. Spielmeyer, W.: Technik der mikroskopischen Untersuchung des Nervensystems, ed. 4, Berlin, Julius Springer, 1930.

No increase of argentophilia or thickening of structures was present. The nuclei of the nerve cells were unstained and visible as light places. The nuclei of the glia cells were in general invisible or only faintly stained in some places (figs. 1 and 2). This normal picture with the Bielschowsky stain in the pieces which were fixed adequately also proves that delay of the fixation for forty and three-fourths hours after death (in the month of August) in this case was not too long a time to give a normal Bielschowsky impregnation.

Fig. 3.—Section from the brain (frontal cortex, inferior part of the third layer) of the same patient as section shown in figure 1. This specimen was incubated in 0.9 per cent saline solution for seventeen hours before fixation in solution of formaldehyde, U.S.P. (1:10). Silver impregnation of the neurofibrils according to Bielschowsky. Soaking change of the Bielschowsky picture; reduced from × 445.

A far different appearance was presented by all the specimens which before fixation were incubated in distilled water, and in 0.3, 0.9 or 20 per cent saline solution (figs. 3 and 4). All the specimens removed from one of these solutions had undergone a characteristic and definite change (figs. 3 and 4). Already at low magnification this change was strikingly apparent in the overimpregnated,

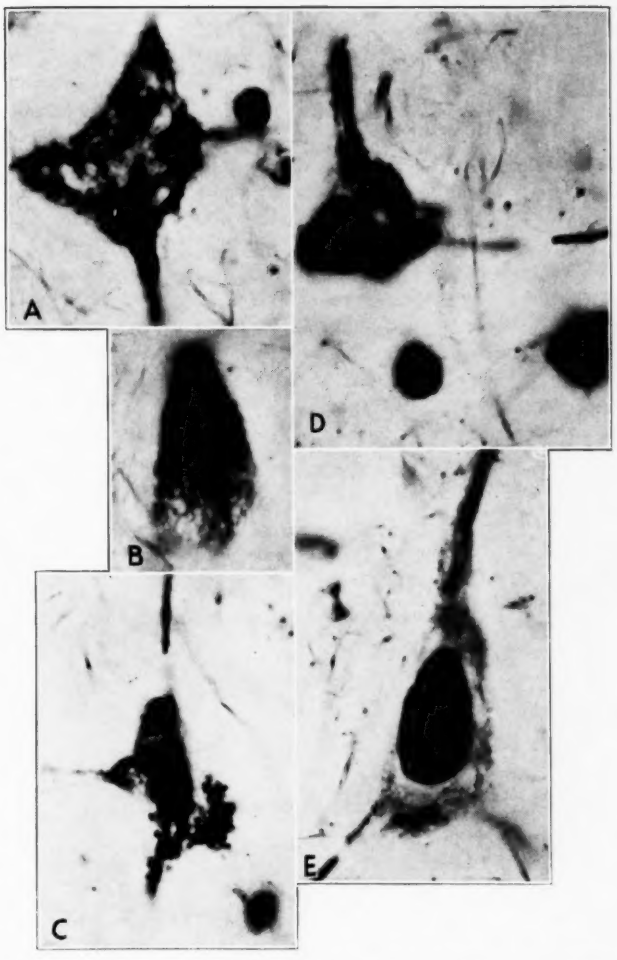

Fig. 4.—Sections from the brain of the same patient as section shown in figure 1. Experimental soaking change of the picture of cortical ganglion cells in Bielschowsky's silver impregnation of neurofibrils, namely, argentophilia, thickening, adhering together (*A*), formation of more or less winding argentophilic strands (*B* and *D*), argentophilia, breaking and crumbling (*C* and *E*) of the intracellular neurofibrils, associated with argentophilia of the nuclei of nerve (*C*, *D* and *E*), and glial (*A*, *C* and *D*) elements. *A*, ganglion cell from the frontal cortex (fifth layer), from a specimen incubated in 0.9 per cent saline solution for seventeen hours before fixation in solution of formaldehyde, U.S.P. (1:10); reduced from × 2,500, oil immersion. *B*, ganglion cell from the frontal cortex (third layer) from a specimen incubated in 0.9 per cent saline solution for seventeen hours before fixation in solution of formaldehyde, U.S.P. (1:10); reduced from × 1,940; oil immersion. *C*, ganglion cell from the frontal cortex (third layer) from a specimen incubated in distilled water for seventeen hours before fixation in solution of formaldehyde, U.S.P. (1:10); reduced from × 1,940, oil immersion. *D*, ganglion cell from the frontal cortex (fifth layer) from a specimen incubated in distilled water for seventeen hours before fixation in solution of formaldehyde, U.S.P. (1:10); reduced from × 1,940, oil immersion. *E*, ganglion cell from the frontal cortex (third layer) from a specimen incubated in 0.9 per cent saline solution for seventeen hours before fixation in solution of formaldehye, U.S.P. (1:10); reduced from × 1,940, oil immersion.

argentophilic, deeply black cell bodies, which they all presented. At higher magnification the intracellular neurofibrils were seen to be thickened and argentophilic. In many instances they adhered together and formed strands of more or less winding course (fig. 4, *A, B, D* and some examples in fig. 3). At other places they were broken into pieces, rods and crumbles (fig. 4, *C* and *E*). No normal intracellular neurofibrils were present in these specimens. Not only the intracellular neurofibrils but also the axis-cylinders in the intracellular tissue were argentophilic (fig. 3), while the ground substance, the ground net itself was even lighter than in the normal specimens (compare fig. 1 and fig. 3). The most striking feature in these specimens, however, was that the nuclei of the large and small ganglion cells, as well as nuclei of glial elements, had become argentophilic and were impregnated with the silver to a deep black color (figs. 3 and 4). It is especially this staining of the nuclei which gives the specimens their characteristic feature conspicuous even at a very low magnification. This becomes clear when one compares the two photomicrographs reproduced together in figure 5. Both are

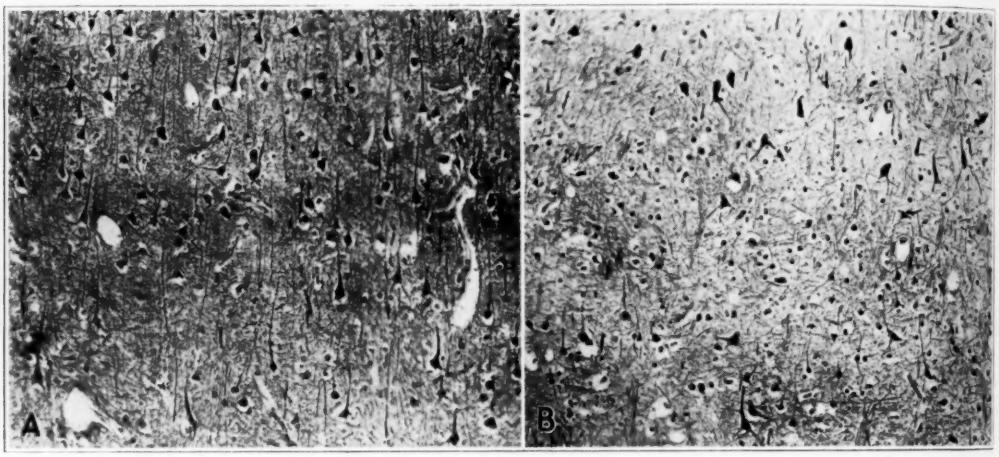

Fig. 5.—Specimens from adjacent areas of the frontal cortex of the brain of the same patient as section shown in figure 1. Both *A* and *B* show the inferior part of the third and the adjacent fourth layer; silver impregnation of the neurofibrils according to Bielschowsky. The two specimens demonstrate the influence of supravital (postmortem) incubation on the Bielschowsky picture at low magnification; reduced from × 155. *A*, section from a specimen which has been fixed in solution of formaldehyde, U.S.P. (1 : 10), immediately after removal from the skull, showing a normal Bielschowsky picture. *B*, section from a specimen incubated in 0.9 per cent saline solution for seventeen hours before fixation in solution of formaldehyde, U.S.P. (1 : 10), showing the soaking change of the Bielschowsky picture.

chosen from places as nearly identical as possible; one from a specimen fixed directly in solution of formaldehyde, the other from a specimen incubated before fixation in 0.9 per cent saline solution. (The appearance of the other specimens incubated in distilled water and in 0.3 per cent saline solution was exactly like that of the one incubated in 0.9 per cent saline solution and not distinguishable from it.) Both figures show the interior part of the third and the fourth layer. In the specimen fixed in solution of formaldehyde (fig. 5 *A*) the neurofibrils of the

large cell bodies are typically stained, while nuclei of all kinds fail to be impregnated. Therefore, there are seen at this magnification the cell bodies and processes of the big cells stained blackish brown and standing out from the ground substance, while the small cells are almost invisible. In the specimen incubated in the saline solution (fig. 5 *B*) many of the big cells appear argentophilic and deep black on a somewhat clearer ground substance, but they seem to be rarefied because the neurofibrils of many of these cell bodies are broken, crumbled or even entirely destroyed. Only the nucleus is left in these cells, and this is darkly impregnated with silver. The chief characteristic of these specimens is that all the nuclei are argentophilic and darkly impregnated with silver, not only the nuclei of the big cells but also those of the small nerve cells and of a number of glial elements. Therefore in these specimens the nuclei stand out and dominate the picture. The small cells become visible on account of their black-stained nuclei.

In regard to the size of the cells in these preparations, it was observed, in confirmation of Weed and McKibben [3] and Fortuyn [5] that though the blocks incubated in distilled water and in 0.3 or 0.9 per cent saline solutions appeared macroscopically markedly swollen, nevertheless on microscopic examination the ganglion cells themselves appeared even shrunken, while only the intercellular substance appeared swollen. In specimens incubated in 20 per cent saline solution the rows of ganglion cells appeared more dense and the intercellular substance was not swollen, but the ganglion cells themselves were also definitely shrunken. In all other respects, especially in respect to general argentophilia of neurofibrils and nuclei as well as to the other neurofibrillar changes, no histologic difference existed between the specimens from the blocks which had been incubated in distilled water or in 0.3, 0.9 or 20 per cent saline solutions.

No argentophilia developed in the specimens incubated in acidulated solution of formaldehyde. The intracellular neurofibrils appeared somewhat denser than normal and adhered together at some places, but they were not argentophilic. Also the nuclei of nerve and glia cells were unstained. The nerve cells were slightly shrunken, but in general the difference from the standard specimen was not marked.

In the specimens incubated in alkaline formaldehyde the gray matter of the cortex was severely damaged. The intracellular neurofibrils and the cells themselves had become entirely unstainable; only some parts of the nuclear walls of some cells were still distinguishable. The nuclei themselves were unstained; no argentophilia was present.

In case 18 examination was performed in the same way and gave exactly the same results in regard to the production of argentophilia and fibrillar alterations by incubation in distilled water or saline solutions. On account of some pathologic features this case will be recorded later under "pathologic cases."

A well known "spontaneous" artefact is Nissl's [45a] so-called "water change" which is found especially in infants but appears also in adults under certain conditions. As in the experimental artefacts, it was found that this change is brought out with the Bielschowsky stain as argentophilia of the nuclei and intracellular neurofibrils, which are thickened, argentophilic, adhering together, broken or crumbled. Formation of rather long continuous strands is an exception, but these strands as well are characterized by a certain crumbled appearance. Figure 6, a photomicrograph

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45a. Nissl, F.: Nervensystem, in Ehrlich, P.; Krause, R.; Mosse, M.; Rosin, H., and Weigert, K.: Enzyklopädie der mikroskopischen Technik, ed. 2, Berlin, Urban & Schwarzenberg, 1910.

of a cell from the third layer of the frontal cortex of a male baby, aged 5 months, shows the features of this water change in the Bielschowsky specimen. The argentophilic nucleus is surrounded by Nissl's retraction space, which obviously indicates the artificial nature of this change, as emphasized also by Scherer.[46] Here also the argentophilia of the nucleus as well as of the two adjacent glia nuclei is the most striking feature. The whole cortex showed uniformly the same change as that represented by this cell. The baby died on Aug. 2, 1933. Autopsy and fixation of the brain in solution of formaldehyde, U.S.P. (1:10), was performed twenty and three-fourths hours later.

Another common postmortem artefact, found also in adults, is a uniform brownish, more or less pale appearance with the Bielschowsky stain, in which the

Fig. 6.—Ganglion cell from the third layer of the frontal cortex of a male baby, aged 5 months; fixation of the brain in solution of formaldehyde, U.S.P. (1:10); silver impregnation according to Bielschowsky. The figure demonstrates the features of Nissl's water change as brought out with the Bielschowsky impregnation; × 1,940.

nuclei appear a rather dark brown and the neurofibrils are crumbled or not impregnated. But no impregnation of strands is obtained in such cases. Only indefinitely outlined, more or less crumbled masses take the stain. This is the usual observation on brains which, as clearly recognized also by other methods, have undergone postmortem change. But the conditions of the occurrence of these spontaneous postmortem artefacts, especially the question of a possible difference in development in edematous and nonedematous brains, will have to be submitted to further study.

46. Scherer, H. J.: Beiträge zur pathologischen Anatomie des Kleinhirns. II. Mitt. Die Erkrankungen des Kleinhirnmarkes und seiner Kerne, insbesondere des Nucleus dentatus, Ztschr. f. d. ges. Neurol. u. Psychiat. **139**:337, 1932.

CONCLUSIONS FROM THE EXPERIMENTAL STUDY

1. Thickening, formation of more or less winding strands and argentophilia of the intracellular neurofibrils can be produced in supravital experiments by incubation of human brain tissue in certain fluids, such as water or saline solutions. This artificial neurofibrillar change may be called incubation or soaking change of neurofibrils.

2. The soaking change of neurofibrils cannot be produced alone but is always accompanied by argentophilia of the nuclei of the nerve cells and glial elements, which normally would not be impregnated in Bielschowsky preparations. Therefore in Bielschowsky preparations the argentophilia of the nuclei serves as an indicator for the neurofibrillar change produced by soaking.

PATHOLOGIC CASES

In the previously mentioned studies on cerebral changes in gastrointestinal infections with terminal cachexia, carried out in collaboration with T. T. Wu, attention was attracted by a considerable change of the neurofibrils in the Bielschowsky picture in some places in a number of brains of patients who died in terminal cachexia with extreme dehydration from dysentery or intestinal tuberculosis, which in one case was complicated by cholera. These brains were well fixed and well preserved, and most of them did not show any appreciable artefacts with other methods. In these brains there were found argentophilia, thickening and formation of all kinds of winding strands, as well as breaking, crumbling or disappearance of the intracellular neurofibrils. Some samples of these cells are reproduced photomicrographically in figure 7. At first these observations were regarded as a confirmation of some of the aforementioned statements in the literature. There seemed to be a parallel between them and the observations of Chanutina [26] and Michailow [27] in cholera, and those of Donaggio [33] who saw fibrillar changes resembling the Alzheimer change in animals starved and frozen to death. This is particularly suggestive in view of the changes in other organs, especially the skin and cornea, which are due to undernutrition and avitaminosis and which are quite common in severe dysentery. Indeed, some of the cells in the specimens observed by Dr. Wu and me showed a great resemblance to the true Alzheimer change. This becomes evident when one compares cell *B* in figure 7 with the cell in the left superior corner in figure 9 of Alzheimer [8b] or with cell *c* in figure 14 of Grünthal [47] or cell *C* of figure 7 with a cell in the left inferior corner of Alzheimer's [8b] figure 8 or with the second from the left in the inferior row of figure 9 of Alzheimer [8b] or with the cell in figure 14, plate 48, of Bielschowsky,[9b] or with cell *d* or *a*

Fig. 7.—Cortical ganglion cells from cases of gastro-intestinal infections with terminal cachexia; fixation in solution of formaldehyde, U.S.P. (1 : 10); silver impregnation of the neurofibrils according to Bielschowsky, showing argentophilia, thickening, adhering together (*A* and *D*), formation of more or less winding argentophilic strands (*B*, *C* and *E*), argentophilia, breaking, crumbling or disappearance (*F*, *G* and *H*) of the intracellular neurofibrils, associated with argentophilia of the nuclei of nervous (*D*, *E*, *F*, *G* and *H*) and glial (*A*, *D*, *E*, *F* and *G*) elements. *A*, *C*, *E*, *F*, *G* and *H*, ganglion cells from the third layer of the frontal cortex of the brain of a man, aged 44, with amebic dysentery (case 1); (*A*, *E*, *F*, *G*, *H*), reduced from × 1,290, oil immersion; *C*, reduced from × 1,940, oil immersion. *B*, ganglion cell from the third layer of the frontal cortex (case 5) (intestinal tuberculosis complicated by Asiatic cholera); reduced from × 1,350, oil immersion. *D*, ganglion cell from the fifth layer of the frontal cortex (case 5); reduced from × 1,200, oil immersion.

in figure 14 of Grünthal.[47] One thing caused hesitancy, however, in considering the change as analogous to a true Alzheimer change of neurofibrils, namely, the fact that nuclei of ganglion cells as well as of surrounding glia cells were stained. They should not be stained in a correct Bielschowsky preparation. This supposition was fully confirmed by experiments already described, which gave as the result that impregnated nuclei are an indicator of a postmortem change which can be produced by soaking the specimens in water or saline solutions before fixation. The similarity of the appearance of the Bielschowsky specimens in the cases of dysentery and intestinal tuberculosis studied by

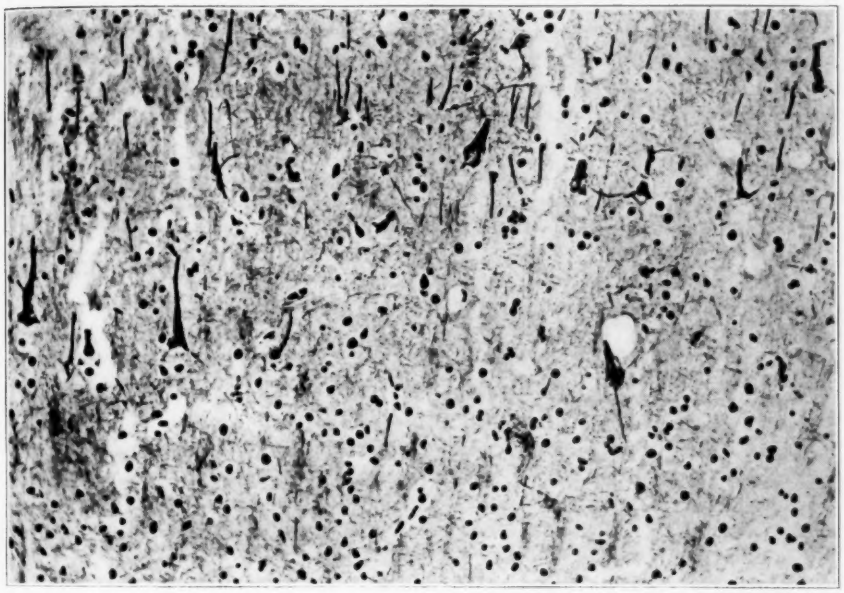

Fig. 8 (case 1).—Section of third layer of the frontal cortex; fixation in solution of formaldehyde, U.S.P. (1:10); silver impregnation of the neurofibrils according to Bielschowsky. The specimen shows argentophilia, thickening, formation of strands of more or less winding course, breaking or disappearance of the intracellular neurofibrils, associated with argentophilia of the nuclei of nervous and glial elements; the changes therefore have to be considered as analogous to the soaking change; reduced from × 234.

Dr. Wu and me with the experimentally produced artificial change is well demonstrated by a comparison of figure 8 (dysentery) with figure 5 *B* (artificial change) and figure 3 (artificial change), or of the cells in figure 7 (dysentery and intestinal tuberculosis) with those in

47. Grünthal, E.: Die pathologische Anatomie der senilen Demenz und der Alzheimerschen Krankheit, in Bumke, O.: Handbuch der Geisteskrankheiten, Berlin, Julius Springer, 1930, p. 638.

figure 4 (artificial change). Therefore at least one component of the neurofibrillar alteration in brains from patients with dysentery must be a postmortem change similar to that produced by soaking specimens in water or saline solutions. On account of this considerable similarity and for the sake of simplicity I should like to use the term "soaking change" for this alteration, but without implying anything in regard to the actual origin of the change. This must be subjected to further analysis. In the following I shall give a brief description of the condition of some brains that showed this change and of some control material. The cases of dysentery and intestinal tuberculosis will be described in extenso in collaboration with Dr. Wu.[48]

CASE 1.—*History.*—A man, aged 44, with chronic amebic dysentery, showed extreme emaciation and dehydration, and terminal cachexia. Agony was prolonged. The heart beat stopped before respiration. Autopsy was performed from fifteen to nineteen hours after death.

Macroscopic Appearances.—The brain was fixed in solution of formaldehyde. Already in the autopsy record "some atrophy of the frontal convolutions of the brain" was noted. Macroscopic inspection of the specimen of the brain revealed an atrophic appearance (Alexander and Wu,[48] figure 1). The convolutions of the entire frontal lobe were very narrow and the sulci much widened. The convolutions of the temporal, parietal and occipital lobe were almost normal; the central convolutions were almost normal but slightly narrower than usual. The macroscopic appearance was that of a true atrophy, but from histologic examination it must be considered as pseudo-atrophy.

Histologic Appearances.—Summary of the Observations in Pyroxylin (Celloidin) Sections, Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Stain: There were sporadic, early small ischemic foci, sporadic disseminated change of the ganglion cells of the cerebral cortex (pyknomorphism and pyknotism), sporadic disseminated deficiency of ganglion cells, glial rosettes and glial stars in the gray matter of the cortex, perivascular glial knots and rims in the white substance.

Stage of Preservation, as Recognized in the Pyroxylin (Celloidin) Sections: With the toluidine blue stain also the normal ganglion cells were stained somewhat darker than usual, apparently on account of the fixation in solution of formaldehyde. No other postmortem artefacts, especially no perinuclear retraction spaces, were present.

Bielschowsky Specimens: At some places there was marked soaking change, with argentophilia, thickening, adhering together, formation of more or less winding strands, and crumbling or destruction of the intracellular neurofibrils, associated with argentophilia of the nuclei of nerve and glial elements (figs. 7 *A, C, E, F, G, H* and 8). At places which were free from the soaking change also, however, the intracellular neurofibrils showed in some instances a tendency to collect along the margin of the cell and along the nucleus (Alexander and Wu[48]).

CASE 2.—*History.*—A man, aged 25, had chronic bacillary dysentery, of the mannite-fermenting group. There were extreme emaciation and dehydration.

48. Alexander, L., and Wu, T. T.: Cerebral Changes in Gastro-Intestinal Infections with Terminal Cachexia: Histopathologic Studies on Dysentery with Comment on Similar Observations in Intestinal Tuberculosis, Arch. Neurol. & Psychiat., to be published.

Fourteen days before death, he became first irrational and later apathetic. There was terminal cachexia. The agony was prolonged. The heart beat stopped before respiration. Autopsy was performed twenty-four and one-half hours after death. The brain was fixed in solution of formaldehyde.

Macroscopic Appearances.—The brain showed slight pseudo-atrophy of the frontal lobes; the convolutions were narrow and the sulci wide. But the pseudo-atrophy was less marked in this case than in case 1; therefore it was not mentioned in the original autopsy record.

Microscopic Appearances.—Summary of the Observations in Pyroxylin Sections Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Method: There were sporadic early ischemic foci, sporadic disseminated change (pyknomorphism) and deficiency of ganglion cells, glial rosettes and glial stars in the gray matter of the cortex and perivascular glial nodules and rims in the white substance.

Stage of Preservation as Recognized in the Pyroxylin Sections: All stains from the pyroxylin sections, especially the Nissl stain, had fully succeeded and did not show any postmortem changes.

Bielschowsky Specimens: At sporadic places, especially along the edges of the blocks, but also in other larger and smaller areas in the interior, a marked typical soaking change of the neurofibrils was found, with argentophilia, thickening, formation of more or less winding strands, breaking or crumbling of the intracellular neurofibrils, associated with argentophilia of nuclei of nerve and glial elements (Alexander and Wu,[48] figure 11). This case is interesting because among larger areas of argentophilia there were small ones as well, represented by only two or three nerve cells which showed the change, but at these places also the nuclei of the adjacent glia cells were argentophilic, thus indicating the nature of the neurofibrillar alteration. All the other cells outside the argentophilic areas gave a normal Bielschowsky impregnation of neurofibrils, with unstained nuclei of ganglion and glia cells.

Case 3.—*History.*—A man, aged 24, had acute bacillary dysentery of the Shiga type. On admission he was weak and undernourished. Mental cloudiness and headache were present in the first week of the disease; later he was dull. He was very miserable. There was some degree of cachexia. There was areflexia in the legs. Death was sudden, under an attack of convulsions with opisthotonos. There was no agony. Autopsy was performed seven and three-fourths hours after death. The brain was fixed in solution of formaldehyde.

Microscopic Appearances.—The brain was available for observation. The convolutions of the frontal lobe were slightly narrower, the gyri somewhat wider than usual, but the condition was not gross enough to be called definitely pseudo-atrophic, at least not without the data of the measurement of the differential ratio of brain volume to skull capacity, which unfortunately were not available in any of these cases.

Microscopic Appearances.—Summary of the Observations in Pyroxylin Sections Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Method: There were sporadic early ischemic foci and some rare perivascular glial rims in the white matter.

Stage of Preservation as Recognized in the Pyroxylin Sections: In the toluidine blue specimens some ganglion cells showed perinuclear retraction spaces (postmortem change), but the number of these cells was small and the degree of the change slight; no other artefacts were present in the Nissl picture.

Bielschowsky Specimens: At various places the characteristic soaking change was found, with argentophilia, thickening, strand formation, and breaking,

crumbling and disappearance of the intracellular neurofibrils associated with argentophilia of the nuclei of the ganglion and glia cells at these places.

CASE 4.—*History.*—A man, aged 49, had chronic bacillary dysentery of the mannite-fermenting group. There were marked dehydration, emaciation, exhaustion, keratomalacia, insomnia, mental dulness, impairment of hearing, and decrease of tendon reflexes. Later the patient was drowsy. There was a terminal cachexia, with prolonged agony. The respiration and heart beat stopped at the same time. Autopsy was performed one week after death. The brain was fixed in solution of formaldehyde. The brain was described as macroscopically unaltered.

Microscopic Appearances.—Summary of the Observations in Pyroxylin Sections Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Method: There were small perivascular foci of ischemic disintegration, disseminated pyknomorphic and pyknotic ganglion cells, marked productive activity of the glia, forming rosettes and stars, clasping ganglion cells and substituting for them as well as forming perivascular glial knots.

Stage of Preservation, as Recognized in the Pyroxylin Sections: The cerebral cortex showed a considerable number of sporadic cells which in the toluidine blue picture presented postmortem changes with extensive perinuclear retraction spaces around a normal but somewhat small and dark nucleus, and protoplasm with a border which was not sharp.

Bielschowsky Specimens: At the outer part of the block there was a characteristic soaking change, with argentophilia of all nuclei and argentophilia, thickening, breaking and for the most part destruction and disappearance of the intracellular neurofibrils, while the interior parts of the block showed a somewhat pale, brownish, uniform but essentially normal Bielschowsky stain.

CASE 5.—*History.*—A man, aged 25, had intestinal and pulmonary tuberculosis, complicated by an acute attack of cholera asiatica. There were extreme emaciation, weakness, dehydration and prostration. The patient was stuporous and lethargic. There was terminal cachexia, with prolonged agony. Autopsy was performed fifteen and three-fourths hours after death. The convolutions of the brain were described as slightly smaller than normal—pseudo-atrophy. Pieces of the brain were fixed in alcohol and solution of formaldehyde.

Microscopic Appearances.—Summary of the Observations in Pyroxylin Sections, Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Method: There were sporadic small early ischemic foci in the cerebral cortex, disseminated regressive change in the ganglion cells (pyknomorphism and pyknotism), glial rosettes, ganglion cell substitutions and perivascular glial knots.

Stage of Preservation as Recognized in the Pyroxylin Sections: There were no postmortem changes, especially not in the Nissl picture.

Bielschowsky Specimens: Characteristic soaking change, with argentophilia and thickening, formation of more or less winding strands (fig. 7 *B* and *D*) and crumbling or disappearance of the intracellular neurofibrils, associated with argentophilia of the nuclei of nerve and glial elements, was found at some places. In one block this soaking change was developed only at its edges, while its center showed normal impregnation (fig. 9). In this case also, however, the same observation as in case 1 could be made, namely, that also in the normally stained parts of the tissue a certain tendency of the neurofibrils to adhere together and to collect along the nucleus and the margin of some cells was recognizable; in these parts, however, many cells showed a normal neurofibrillar picture.

Fig. 9 (case 5).—Section from the frontal cortex (third to sixth layers), from a specimen fixed in solution of formaldehyde, U.S.P. (1 : 10) ; silver impregnation of the neurofibrils according to Bielschowsky. The edge of the specimen (right half of the picture) shows a marked soaking change, its central part (left half of the picture) shows a normal Bielschowsky picture ; reduced from × 104.

Case 6.—*History.*—A girl, aged 17, had intestinal and pulmonary tuberculosis. She was extremely undernourished and emaciated, and had ascites and anasarca. There were headache, double vision and apathy. Six days before death there occurred one short attack of convulsions, with unconsciousness. There was terminal cachexia with prolonged agony. Autopsy was performed twenty-four hours after death. Slight dilatation of the ventricles was noted. The brain was fixed in solution of formaldehyde, some pieces being placed in alcohol.

Macroscopic Appearances.—Pseudo-atrophy of the anterior parts of the frontal lobe was present; here the gyri were very narrow, the sulci very wide (Alexander and Wu,[48] fig. 16 *B*).

Microscopic Appearances.—Summary of the Observations in Pyroxylin Sections Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Method: There were few small early ischemic foci, sporadic disseminated pyknomorphic ganglion cells in the cerebral cortex, glial rosettes, stars and some perivascular increase of glia.

Stage of Preservation as Recognizable in the Pyroxylin Sections: A small number of sporadic cortical ganglion cells showed in the Nissl picture a somewhat uniform stain of the protoplasm and a perinuclear retraction space; there was slight postmortem change.

Bielschowsky Specimens: There were larger and smaller areas of typical soaking change, with argentophilia, thickening, formation of strands and crumbling and disappearance of the intracellular neurofibrils, associated with argentophilia of the nuclei of nerve and glial elements. The areas which did not show the soaking change gave an entirely normal neurofibrillar picture, without any tendency of the neurofibrils to adhere together or to collect in certain parts of the cells.

Case 7.—*History.*—A man, aged 26, had intestinal and pulmonary tuberculosis and was emaciated. He was delirious for about one week before death. There was terminal cachexia with prolonged agony. Autopsy was performed twenty-four hours and forty minutes after death. The brain was fixed in solution of formaldehyde. Dilatation of the ventricles was noted.

Macroscopic Appearances.—The brain was available for observation. There was pseudo-atrophy of the frontal and of the occipital lobe (Alexander and Wu,[48] fig. 16 *A*). Marked dilatation of the lateral ventricles was present.

Microscopic Appearances.—Summary of the Observations in Pyroxylin Sections Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's method: There were slight fibrosis of the meninges; sporadic small early ischemic foci in the gray matter of the cerebral cortex; multiple early foci of softening, but without dissolution of the tissue, in the white matter; disseminated sporadic pyknomorphic ganglion cells in the cortex; glial rosettes; glial stars and sporadic perivascular increase of glia.

Stage of Preservation, as Recognizable in the Pyroxylin Sections: In specimens stained with toluidine blue one or the other cortical ganglion cell showed a nucleus that was slightly too dark or a perinuclear retraction space. However, the stage of preservation could in general be considered as satisfactory.

Bielschowsky Specimens: Regions were found in which the typical soaking change had developed: argentophilia, thickening, strand formation and crumbling or disappearance of the intracellular neurofibrils, associated with argentophilia of the nuclei of nerve and glial elements. Other regions showed an entirely normal neurofibrillar picture.

CASE 8.—*History.*—A man, aged 22, with pulmonary and intestinal tuberculosis, was emaciated and had anasarca. Mentally he was drowsy. There was terminal cachexia, with prolonged agony. Autosy was performed six days after death. The brain was fixed in solution of formaldehyde.

Macroscopic Appearances.—There was no pseudo-atrophy or dilatation of the ventricles.

Microscopic Appearances.—Summary of the Observations in Pyroxylin Sections Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Method: There were small ischemic foci, disseminated pyknomorphic cells, glial rosettes and glial stars.

Stage of Preservation as Recognizable in Pyroxylin Sections: There were definite postmortem changes, especially in specimens stained with toluidine blue, many of the normal as well as many of the pathologic cells showing marked perinuclear retraction spaces and in many instances vacuolation of the pale protoplasm.

Bielschowsky Specimens: In some regions the typical soaking change had developed, with thickening, strand formation, argentophilia, and crumbling and disappearance of the intracellular neurofibrils, associated with argentophilia of the nuclei of nerve cells and glia. But in other regions the neurofibrillar impregnation was fully normal, or, in some instances, normal but somewhat pale. It was noteworthy that in this case also at places which showed normal or only slightly pale neurofibrillar impregnation some of the nuclei of the ganglion cells and of the adjacent glial elements had a tendency to take on a brownish stain similar to that of the neurofibrils.

CASE 9.—*History.*—A man, aged 23, with intestinal and pulmonary tuberculosis had extreme emaciation and prostration, with dehydration. There was terminal cachexia, with prolonged agony. Autopsy was performed five days after death. The brain was described as anemic.

Macroscopic Appearances.—The brain, fixed in solution of formaldehyde, was available for examination. It showed no pseudo-atrophy and no dilatation of the ventricles.

Microscopic Appearances.—Summary of the Observations in Pyroxylin Sections Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Method: There were sporadic small early ischemic foci, disseminated pyknomorphic cells, and glial rosettes.

Stage of Preservation, as Recognizable in Pyroxylin Sections: The Nissl picture was astonishingly well preserved, if one considers the long time between death and fixation. However, the sections showed definite postmortem changes: In the central parts of the white matter of the convolutions round holes due to putrefaction were visible, and a number of ganglion cells, scattered throughout the cortex, showed perinuclear retraction spaces and vacuolation.

Bielschowsky Specimens: Only one feature of the soaking change had developed completely at some places, namely, argentophilia of the nuclei of ganglion cells and glial elements; there the neurofibrils were thickened, with formation of short fragmented strands and rods, and crumbling or even disappearance, but with less argentophilia than in other cases. The nonargentophilic regions, however, also presented a much altered Bielschowsky picture in this case. They showed a brownish appearance, in which also the nuclei stood out in a somewhat darker brownish shade, while the neurofibrils adhered together, forming crumbled indistinct masses, especially along the margins of the cells, or had entirely disappeared.

From this report it is seen that the four cases of dysentery and the five cases of intestinal tuberculosis (complicated in one case by cholera asiatica) studied with Bielschowsky's method showed at some places the alteration of the Bielschowsky picture described as soaking change. In only one case was it less marked, though present.

The following report of cases deals with other material examined.

Bronchopneumonia.—Case 10.—*History.*—A boy, aged 8 months, had bronchopneumonia with a very acute course of five days. Agony was short. Autopsy was performed on the day of death, probably six hours after death. The brain was fixed in solution of formaldehyde, some pieces being placed in alcohol.

Macroscopic Appearances.—The brain revealed no changes, especially no pseudo-atrophy.

Microscopic Appearances.—Stage of Preservation as Recognizable in Pyroxylin Sections: In the Nissl picture the ganglion cells presented Fortuyn's inflated type, with pale protoplasm and pale, somewhat indistinct Nissl bodies. There were no other alterations.

Bielschowsky Specimens: These were entirely normal.

Case 11.—*History.*—A boy, aged 16 months, had scarlet fever with bronchopneumonia of fourteen days' duration. The course was acute, and agony was short. Autopsy was performed nineteen and one-fourth hours after death. The brain was fixed in solution of formaldehyde.

Macroscopic Appearances.—The brain revealed no changes, especially no pseudo-atrophy.

Microscopic Appearances.—Stage of Preservation as Recognizable in Pyroxylin Sections Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Method: The specimens stained with toluidine blue showed typical water change (Nissl).

Bielschowsky Specimens: At some places throughout the brain the nuclei were dark and argentophilic. In most of the ganglion cells neurofibrils had crumbled or disappeared; in rare instances short, crumbled, argentophilic rods were visible. There was no formation of strands. Some of the Betz giant cells alone resisted postmortem destruction and showed an almost normal neurofibrillar picture, but also with dark-stained nuclei.

Acute Nephritis.—Case 12.—*History.*—A man, aged 21, had acute nephritis with anasarca of about one month's duration. Agony was short. Autopsy was performed twelve hours and thirty minutes after death. The brain was normal macroscopically, especially it was not swollen; it appeared anemic. It was fixed in solution of formaldehyde, some pieces being placed in alcohol.

Macroscopic Appearances.—The brain revealed no pseudo-atrophy.

Microscopic Appearances.—Stage of Preservation as Recognizable in Pyroxylin Sections Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Method: There was no postmortem change.

Bielschowsky Specimens: The stain was somewhat uniform but essentially normal; especially, no soaking change and no neurofibrillar alteration were apparent.

Malaria.—Case 13.—*History.*—A man, aged 30, had pernicious (subtertian) malaria of peracute course. Death occurred after three days with agony of considerable duration—about twelve hours. Autopsy was performed thirteen days after death. The brain was soft; it was fixed in solution of formaldehyde.

Macroscopic Appearances.—The brain was available for examination. There was no pseudo-atrophy.

Microscopic Appearances.—Stage of Preservation as Recognizable in Pyroxylin (Celloidin) Sections Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Method: In specimens stained with toluidine blue definite postmortem changes were present—nuclei small and dark, protoplasm faintly stained, vacuolated, perinuclear retraction spaces and indistinct outer cell borders.

Bielschowsky Specimens: The tissues were pale and somewhat uniform, stained in a grayish-brown shade. No argentophilia had developed; all the nuclei were clear and unstained, and in many ganglion cells faintly stained, but distinct neurofibrils of normal structure and distribution were visible.

Septicemia.—Case 14.—*History.*—A man, aged 28, had a Streptococcus haemolyticus septicemia from a tonsillar abscess, with a very acute course of fourteen days' duration. Death occurred from circulatory failure, with short agony. Autopsy was performed eighteen hours after death. The meninges were slightly thickened; the brain was macroscopically normal, and was fixed in solution of formaldehyde.

Macroscopic Appearances.—The brain showed no pseudo-atrophy.

Microscopic Appearances.—Stage of Preservation as Recognized in Pyroxylin Sections Stained with Toluidine Blue, Hematoxylin-Eosin and van Gieson's Method: With toluidine blue, ganglion cells in general were stained somewhat darker than usual, apparently owing to the fixation in the solution of formaldehyde. No other artefacts were present in the Nissl picture.

Bielschowsky Specimens: The staining was entirely normal.

Case 15.—*History.*—A woman, aged 40, had acute Streptococcus haemolyticus tonsillitis, pharyngitis and septicemia of one week's duration, complicated by a miscarriage during the course of the disease, and later by bronchopneumonia. Agony was short (a few hours). Autopsy was performed one hour and ten minutes after death. The brain, macroscopically normal, was fixed in solution of formaldehyde.

Macroscopic Appearances.—The brain, which was available for examination, showed no pseudo-atrophy.

Microscopic Appearances.—Stage of Preservation as Recognized in Pyroxylin Sections: There were no artefacts, especially none in toluidine blue specimens.

Bielschowsky Specimens: The staining was entirely normal.

Case 16.—*History.*—A woman, aged 34, had acute Streptococcus haemolyticus septicemia, peritonitis and cellulitis of the femoral region from endometritis after hysterotomy at four months of pregnancy, which was performed on account of an eclamptic condition that had also caused foci of softening in the brain with clinical focal symptoms. Two weeks before admission there was edema; four days before admission there was a sudden onset of eclamptic convulsions. Admission was on Nov. 3, 1927, operation on November 4. Fever was present on admission and became septic in type on November 7. Death occurred on November 28, at 1:25 p. m. Agony was prolonged. The heart beat stopped before respiration. Autopsy was performed on the day of death, probably from two to seven hours after death. The brain was fixed in solution of formaldehyde.

Macroscopic Appearances.—The brain showed white thrombi in some pial veins. No other alteration of the external surface was detected, especially no pseudo-atrophy.

Microscopic Appearances.—There were a great number of circulatory foci of all sorts and degrees: early small ischemic foci, extensive ischemic foci, ischemic necrosis, and incomplete and complete softening. Outside the foci sporadic disseminated damaged ganglion cells were present throughout the cortex, mostly an ischemic change, but also pyknomorphism and pyknotism. A great number of cortical ganglion cells outside the foci, however, were normal. There were glial stars, glial rosettes and perivascular glial knots.

Stage of Preservation as Recognized in Pyroxylin Sections: There were no artefacts, especially none in the Nissl picture.

Bielschowsky Specimens: Outside the foci there was entirely normal neurofibrillar impregnation.

CASE 17.—*History.*—A man, aged 55, a morphine addict, had a chronic septic condition arising from many extensive infected ulcers at the site of injections, with severe emaciation and terminal cachexia. Agony was prolonged (two days).

Fig. 10 (case 18).—Ganglion cell from the third layer of the frontal cortex. Fixation in solution of formaldehyde, U.S.P. (1:10); silver impregnation of the neurofibrils according to Bielschowsky. This cell shows thickening, argentophilia and strand formation of the intracellular neurofibrils associated with argentophilia of the nucleus (soaking change); × 1,940, oil immersion.

Autopsy was performed two days after death. The brain was fixed in solution of formaldehyde, pieces being placed in alcohol. The brain was described as macroscopically normal, but the specimen was not available for examination.

Microscopic Appearances.—Stage of Preservation as Recognizable from Pyroxylin Specimens: In the Nissl specimens there was typical water change (Nissl).

Bielschowsky Specimens: Throughout the pieces examined the nuclei of ganglion and glia cells were dark and argentophilic, while the intracellular neurofibrils were destroyed or formed argentophilic fragments, crumbled rods and short strands of crumbled appearance.

Kala-Azar.—CASE 18.—*History.*—A boy, aged 6, had kala-azar, with marked generalized nutritional anasarca. He was undernourished, but not cachectic; he

was clear mentally. Death occurred suddenly after the intravenous injection of acacia; there was no agony. Autopsy was performed forty-one hours after death.

Macroscopic Appearances.—The brain was examined in the fresh unfixed state, as removed at autopsy. There was no macroscopic change. (Unfortunately also in this brain the difference between the capacity of the skull and volume of the brain was not measured according to Reichardt's and Rudolph's method.[48a])

Behavior of the Fresh Brain Tissue Toward Different Fluids.—By distributing pieces of this brain in the various fluids in the same way as in case 1, some remarkable differences were shown. Not only the pieces placed in distilled water and the 0.3 and 0.9 per cent saline solutions, but also the pieces put into solution of formaldehyde showed remarkable swelling. The pieces in 20 per cent saline solution and the pieces in acidulated solution of formaldehyde (10 drops of concentrated glacial acetic acid in 65 cc. of solution of formaldehyde, U.S.P. [1:10]), kept their shape and size fairly well. Certainly no shrinking occurred. The pieces in 20 per cent saline solution were possibly slightly swollen. The pieces in acidulated solution of formaldehyde and in 20 per cent saline solution were the best hardened. The pieces in alkaline formaldehyde (10 drops of sodium hydroxide in 65 cc. of solution of formaldehyde, U.S.P. [1:10]) showed no change in shape or size, but became smooth in consistency.

Bielschowsky Specimens: The pieces soaked in water and in the saline solutions showed in general the same features as in case 1. The material originally fixed in solution of formaldehyde showed some cells with thickening, argentophilia and strand formation or breaking and crumbling of the intracellular neurofibrils, as well as argentophilia of the nucleus (soaking change). Figure 10 shows one of these cells. They were found in abundance in the specimens soaked in water or saline solution, but were rare in specimens fixed in solution of formaldehyde, in which one had to look especially for them. Still less were found in pieces that had been fixed in acidulated solution of formaldehyde; in these a much larger number of cells was impregnated normally than in the specimens hardened in solution of formaldehyde, U.S.P. (1:10).

This case shows that a certain parallel between argentophilia and postmortem swelling exists, and that this postmortem swelling in different fluids may be different in individual cases.

Stage of Preservation as Recognized in Pyroxylin Sections: In toluidine blue specimens from material fixed in solution of formaldehyde, U.S.P. (1:10), most of the cells had a normal configuration, but some sporadic ganglion cells showed perinuclear retraction spaces or vacuolation of the protoplasm.

THE "SOAKING CHANGE" OF THE INTRACELLULAR NEUROFIBRILS

The occurrence of the previously described soaking change of the Bielschowsky picture in different pathologic cases seems to be related to various factors in these cases, such as edematous conditions or severe emaciation and dehydration, terminal cachexia, long agony and postmortem changes. It might be noted, however, that the soaking change may also be present when other methods, especially the Nissl stain, do not show postmortem changes, and may also be absent when

48a. Rudolph, O.: Untersuchungen über Hirngewicht, Hirnvolumen und Schädelkapazität, Beitr. z. path. Anat. u. z. allg. Path. **58**:48, 1914.

other methods reveal definite postmortem changes. At least one factor in its production, however, seems to occur post mortem, since the change appears to become at least manifested during fixation. This is suggested by the fact that in some cases the change is especially developed along the edges of some blocks (fig. 9). But the alteration of the brain material which might serve to prepare for this peculiar action of the fixation fluid,[49] namely, to produce a soaking change of the tissue, could not be traced to its ultimate cause. It was not possible at the time to determine the special significance of the various probable causal components—edematous conditions, emaciation, dehydration, terminal cachexia, agony and postmortem changes—and the way in which they might interfere with each other. This must be left for further study.

One definite result is brought out by these studies, namely, the fact that this soaking change is always characterized by argentophilia of nuclei of nerve and glia cells, which should not be impregnated in a correct Bielschowsky stain.

This has the practical consequence that every Bielschowsky specimen which shows neurofibrillar alterations together with argentophilia of nuclei should be interpreted only with the greatest precaution, and its relationship to systemic diseases and postmortem conditions should be considered.

This fact and its importance for the interpretation of a neurofibrillar picture has hitherto not been noted; no attention was paid to it by some authors who described the atypical (nonsenile or presenile) occurrence of neurofibrillar alterations. The photographs (figs. 9, 10, 11, 12 and 13) of Van Bogaert and Bertrand [23] show definite argentophilia (dark silver impregnation) of the nuclei of ganglion and glia cells, and some cells in their figures 10, 11 and 13 also seem to contain perinuclear retraction spaces. Also figures 2, 3 and 5 of Malamud and Lowenberg,[25] from their atypical as well as from their senile case, show definite argentophilia of the nuclei. Therefore the neurofibrillar alteration in these cases must be considered not as comparable to the genuine Alzheimer change but as a soaking change, which might even conceal a genuine neurofibrillar change if it were present.

PSEUDO-ATROPHY OF THE BRAIN IN SEVERE SYSTEMIC DISEASES

In the previously described cases of severe gastro-intestinal infections with terminal cachexia, another feature also attracted attention, namely, the peculiar macroscopic condition described as pseudo-atrophy. That is the occurrence of narrow convolutions and wide sulci, espe-

49. That certain fluids may produce argentophilia has been recently demonstrated by van Herweden (Argyrophile Netze und Fibrillen in nicht organisierten Membranen, Protoplasma **19**:330, 1933) who found argentophilic nets and fibrils in inorganic membranes, which develop in compound solution of cresol (5 per cent).

cially in the frontal lobe, sometimes associated also with slight dilatation of the ventricles.

I can say only that this atrophic condition is at first independent of meningitic or meningofibrotic conditions, especially in tuberculosis, and furthermore cannot be caused by the early ischemic foci (which did not yet cause dissolution of the tissue substance) and also not by the slight disseminated parenchymatous change, which was not severe enough to cause any gross alteration, especially no disarrangement of the cortical architecture.

Therefore some other unknown substance, in addition to the ganglion cells, must have been altered in these brains. A certain amount of probability exists that dehydration may play a rôle in the production of this "emaciation" or pseudo-atrophy of the brain. Especially the fact that in severely dehydrated children depression of the fontanel may be noted, as in case 25 of the anatomic investigation carried on with Dr. Wu, points in this direction.

Similar pseudo-atrophic conditions of the brain, even proved by the measurement of the difference between skull capacity and brain volume, have been described by Apelt [50] in systemic disease with chronic course and general cachexia. Apelt considered this to be a decrease of "brain mass," but gave no further specification in detail. Recently, "atrophy" of the brain, especially of the frontal lobe, has been reported in pellagra by Wyjasnowsky.[51] But I am inclined to consider this as a pseudo-atrophy in line with the observations in the cases of dysentery and tuberculosis studied.

The nature of these conditions needs physicochemical investigation. One of the next steps in this direction might be to determine the water content of the brain in various systemic diseases and conditions, if only to rule out this possibility. Apelt [2] tried to determine differences in the water content of the brain, but failed. With the method of drying and weighing small pieces he was unable to determine any difference between brains which were swollen and those which were not. He was inclined to believe with Reichardt that in cases of swelling of the brain the water is bound in a special colloidal form. But one of the difficulties may also be that the normal brain, according to Reichardt, is already rich in water (70 per cent for the white and 83 per cent for the gray matter). Therefore a change in the amount and distribution of this fluid content may be significant, yet not great enough

50. Apelt, F.: Der Wert von Schädelkapazitätsmessungen und vergleichenden Hirngewichtsbestimmungen für die innere Medizin und die Neurologie, Deutsche Ztschr. f. Nervenh. **35**:306, 1908.

51. Wyjasnowsky, A. J.: Ueber Pellagra-Psychosen (klinische Bemerkungen und Beobachtungen), Ztschr. f. d. ges. Neurol. u. Psychiat. **144**:388, 1933.

to be measurable with the scale or to be beyond the limits of error. However, these difficulties may not be insurmountable; further study and inquiry may reveal some points of interest.

SUMMARY

1. Argentophilia, thickening, formation of more or less winding strands and crumbling and destruction of the intracellular neurofibrils demonstrated in Bielschowsky's silver impregnation can be produced in postmortem (supravital) experiments by soaking fresh human brain tissue in certain fluids, such as distilled water and saline solutions. This soaking change of the neurofibrils is always accompanied in the same tissue by argentophilia of the nuclei of nerve and glia cells. Since these nuclei are pale in normal Bielschowsky specimens, they are an easily recognizable indicator for this soaking change of the neurofibrils. Argentophilia of these nuclei is also found in a spontaneous artefact, the so-called "water change," which occurs especially in children.

2. Soaking change of the intracellular neurofibrils, associated with the characteristic argentophilia of the nuclei, was found in some cases of severe gastro-intestinal infections with terminal cachexia, such as dysentery, intestinal tuberculosis and intestinal tuberculosis combined with cholera asiatica. This change was not necessarily associated with any other postmortem change in these cases.

3. The soaking change may be due to various factors, such as edematous conditions, emaciation, dehydration, terminal cachexia, long agony and postmortem changes. Its exact nature must be subjected to further study.

4. A reliable indicator is given for the recognition of this soaking change of the neurofibrils, namely, argentophilia of the nuclei in the same specimen.

5. Therefore any specimen showing neurofibrillar changes together with argentophilia of the nuclei should be interpreted with great precaution, and its relation to the aforementioned factors should be considered. This precaution has not been observed by some authors who described cases of the atypical (nonsenile or presenile) occurrence of neurofibrillar alterations.

6. A peculiar alteration of the macroscopic appearance (pseudoatrophy) was also noted in the brains in some of the cases observed. Its nature has not yet been determined. Its possible relationship to dehydration is discussed.

THE PREMOTOR AREA

ITS RELATION TO SPASTICITY AND FLACCIDITY IN MAN

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Recent experimental investigations have demonstrated that the cortical motor zone may be divided physiologically into two areas: the anterior or premotor area, corresponding to area 6 of Brodmann and area F B of von Economo, and the posterior or the motor area, corresponding to area 4 of Brodmann and F A of von Economo (fig. 1). In their experiments with cortical ablation in primates, Kennard and Fulton [1] observed that a lesion restricted to area 4 resulted in a contralateral flaccid hemiplegia. Spasticity appeared only when area 6 was removed. These investigators concluded that in primates spasticity is present after the premotor area has been ablated and fails to appear if the area remains intact. Subsequently Kennard, Viets and Fulton [2] reported a case in which a cystic astrocytoma restricted to the right premotor area and in the vicinity of the arm and leg led to focal seizures of the left side of the body, progressive rigidity, awkwardness and stiffness of the left arm, forced grasping, increased reflexes and vasomotor disturbances. The removal of the neoplasm led to recovery and to a temporary "complete *flaccid* paralysis." On the twenty-fifth day after operation persistent spasticity of moderate degree appeared. Kennard, Viets and Fulton concluded from their study of the case that in

> . . . premotor lesions, awkwardness, *spasticity* and increase of tendon reflexes appear early, before the onset of motor weakness; whereas in lesions of the motor area, weakness begins early, reflexes are at first depressed, and *spasticity*, if present, appears late. . . . Acute injuries or rapidly expanding lesions of the motor area produce *flaccid* paralysis and generalized depression of the reflexes.

The present investigation was undertaken with a view toward correlating by clinicopathologic studies the relation of spasticity to the pre-

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From the Neuropathological Laboratory, Montefiore Hospital.

1. Kennard, M. A., and Fulton, J. F.: The Localizing Significance of Spasticity, Reflex Grasping and the Signs of Babinski and Rossolimo, Brain **56:** 213, 1933.

2. Kennard, M. A.; Viets, H. R., and Fulton, J. F.: The Syndrome of the Premotor Cortex in Man: Impairment of Skilled Movements, Forced Grasping, Spasticity, and Vasomotor Disturbance, Brain **57:**69, 1934.

motor area in cerebrovascular disease in man. As the closure of the middle cerebral artery plays by far the most common rôle in spastic hemiplegia, a group of about fifty cases with complete occlusion of this vessel were selected. In the complete occlusions of the middle cerebral artery the lower two thirds of the premotor area was involved. This

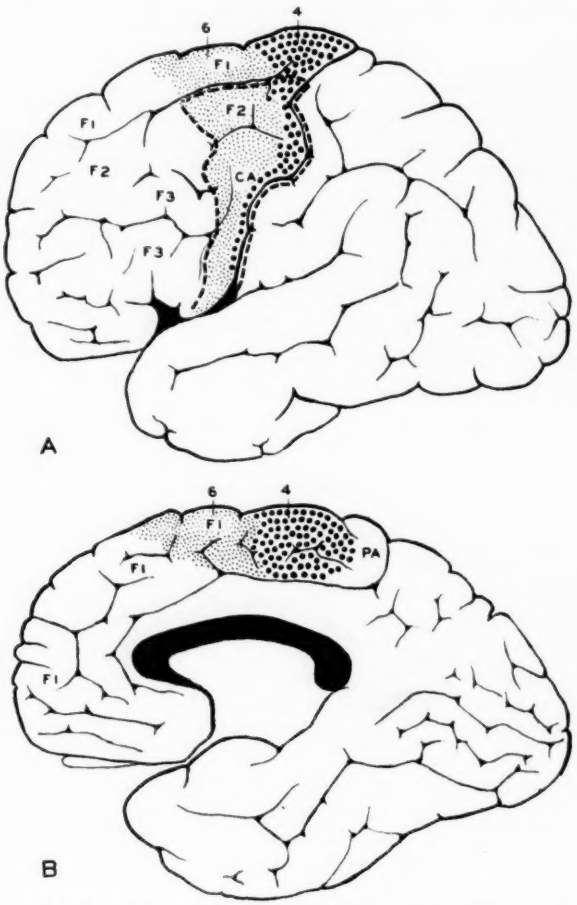

Fig. 1.—*A*, lateral view of the brain, showing the premotor and motor zones (areas 6 and 4 of Brodmann). The premotor area (area 6 of Brodmann) is formed by the posterior part of the first frontal (*F1*), second frontal (*F2*) and the lower part of the precentral (*CA*) convolutions. The middle cerebral artery supplies the premotor area formed by the second frontal and part of the precentral convolutions. The area enclosed by the heavy dashes is the premotor and motor areas involved in our cases of flaccid hemiplegia. This is the area supplied by the middle cerebral artery. The superior lateral and mesial surfaces of the premotor area formed by the posterior part of the first frontal convolution (*A* and *B*) and the anterior part of the paracentral lobule (*B*) are supplied by the anterior cerebral artery. These areas were not implicated in our series. *B*, mesial view of the brain, showing the premotor and motor areas. *PA* indicates the paracentral lobule.

corresponds to the posterior segment of the second frontal convolution and the lower two thirds of the precentral gyrus, indicated by *CA* in figure 1 *A*. The upper third and the mesial part of the premotor area represented in the first frontal convolution and the anterior part of the paracentral lobule (fig. 1 *B*) are supplied by the anterior cerebral artery. In the series of fifty cases of complete thrombosis of the middle cerebral

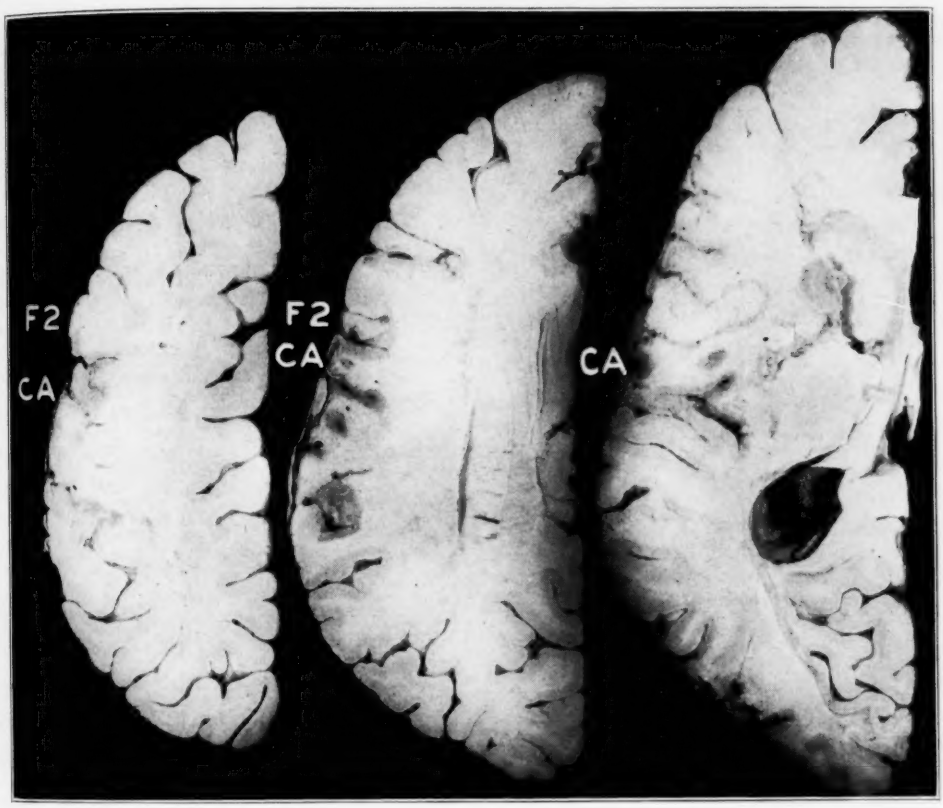

Fig. 2 (case 1).—Destruction of the convolutions of the right hemisphere irrigated by the right middle cerebral artery. Notice the destruction of the second frontal and precentral convolutions in three successive horizontal segments about 1 cm. apart.

artery, three were cases of flaccid hemiplegia, and the rest, of spastic hemiplegia. Three additional cases of flaccidity due to partial occlusion of the middle cerebral artery were included in the group. The degree of premotor implication in the six cases of flaccid hemiplegia was compared with the extent seen in the cases of spastic hemiplegia. The shortest acceptable period for the duration of flaccidity was taken as eight weeks.

We believe that if the situation in man were strictly analogous to that in the other primates one should not expect to find involvement of the premotor area or at least little implication of it in flaccid hemiplegia.

REPORT OF CASES

CASE 1.—*Complete closure of the right middle cerebral artery. Flaccid hemiplegia of two months' duration. Destruction of the lower two thirds of the premotor area.*

Fig. 3 (case 2).—Destruction of the convolutions of the right hemisphere along the distribution of the right middle cerebral artery. Notice the destruction of the second frontal and the precentral convolutions which form the premotor area. (Part of the area marked *CA*, missing in this section, was removed for microscopic study.)

History.—A. A., a woman, aged 45, was admitted to the Montefiore Hospital on Dec. 3, 1931, with a history of an acute cerebral episode with coma in October 1931 which resulted in left flaccid hemiplegia.

Neurologic examination disclosed complete left flaccid hemiplegia, hyperreflexia, absence of the left abdominal reflexes, positive Babinski and Mendel-Bechterew responses and left sensory disturbances for all of the modalities. The patient died following acute coronary thrombosis on December 17. The flaccid hemiplegia lasted two months.

Autopsy.—There was complete thrombosis of the right middle cerebral artery, with softening of the right second and third frontal, precentral, postcentral, parietal, angular, supramarginal and first temporal convolutions, as well as of the subcortical areas supplied by this artery (fig. 2). The lower two thirds of the premotor area was completely destroyed.

CASE 2.—*Complete occlusion of the right middle cerebral artery. Flaccid hemiplegia of three months' duration. Involvement of the lower two thirds of the premotor area.*

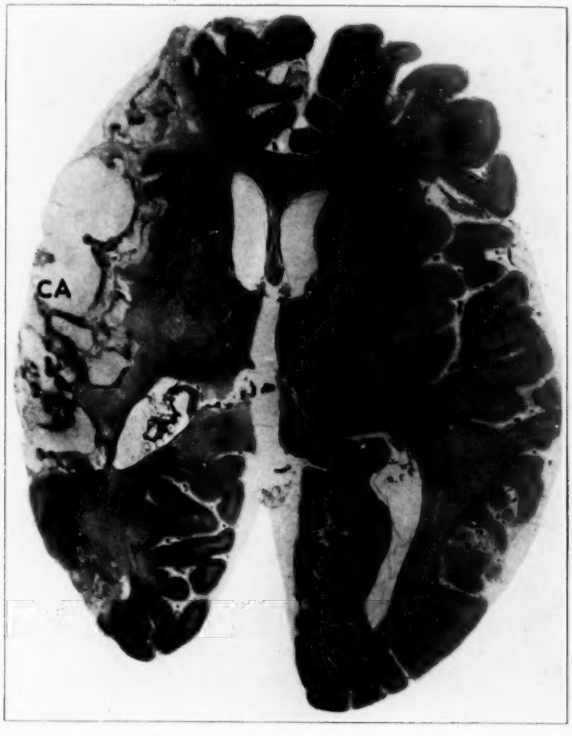

Fig. 4 (case 3).—Destruction of the convolutions of the right hemisphere along the distribution of the right middle cerebral artery, with involvement of the second frontal and precentral convolutions which enter into the formation of the premotor area.

History.—J. P., a man, aged 65, was admitted to the Montefiore Hospital on Sept. 24, 1931, with a history of two vascular insults in the right hemisphere in June and July 1931, respectively. Following the last episode the patient remained bedridden until death.

Neurologic examination disclosed left flaccid hemiplegia, with complete loss of power in the left extremities. There was moderate left hyperreflexia. The abdominal reflexes were present. Neither the Babinski response nor its confirmatories were elicited. There was impairment of all of the forms of sensation on the left. The patient died on October 8. The flaccid hemiplegia lasted three months.

Autopsy.—There was a large area of softening along the distribution of the right middle cerebral artery, with destruction of the right second and third frontal, precentral, postcentral, inferior parietal, angular, supramarginal and insular convolutions and the globus pallidus, putamen and the anterior two thirds of the capsule (fig. 3). The premotor area, with the exception of the first frontal convolution, was thoroughly destroyed.

CASE 3.—*Complete closure of the right middle cerebral artery. Left flaccid hemiplegia of three months' duration. Involvement of the lower two thirds of the premotor area.*

Fig. 5 (case 4).—Incomplete occlusion of the left middle cerebral artery with destruction of the fibers from the second frontal and precentral convolutions which form the premotor area.

History.—C. A., a man, aged 65, was admitted to the Montefiore Hospital with a history of hypertension for several years. A complete left-sided paralysis appeared in February 1931.

Neurologic examination revealed complete left flaccid hemiplegia with all the pyramidal tract signs, impairment of all of the modalities of sensation on the left and left homonymous hemianopia. The flaccid hemiplegia persisted for three months, until the patient died on May 15.

Autopsy.—There was complete closure of the right middle cerebral artery, with softening of the following convolutions: second and third frontal, precentral,

opercular, insular, postcentral, superior parietal, inferior parietal, precuneus, supramarginal, angular, first and second temporal and part of the third temporal (fig. 4). The right internal capsule and part of the subcortical structures supplied by this vessel were also involved.

CASE 4.—*Incomplete occlusion of the left middle cerebral artery. Flaccid hemiplegia of five months' duration. Partial involvement of the lower two thirds of the premotor area.*

History.—E. T., a woman, aged 60, was admitted to the Montefiore Hospital on March 18, 1932, with a history of right-sided paralysis and loss of speech in December 1930.

Neurologic examination disclosed global aphasia and complete flaccid paralysis of the right upper extremity, with mild paresis of the right lower extremity, trophedema of the right upper extremity and generalized right hyperreflexia with positive Babinski and Hoffmann responses. Accurate sensory evaluation was impossible because of the aphasia. The flaccid hemiplegia lasted five months. Death occurred suddenly on May 2, 1932.

Autopsy.—There was incomplete occlusion of the left middle cerebral artery, with involvement of parts of the left second and third frontal, precentral and parietal convolutions, as well as of the neostriatum and the internal capsule. The white matter was more involved than the gray. Microscopic examination, however, showed that the gray matter of the premotor area was not spared (fig. 5). There were distortion in the cyto-architectural layers, areas of destruction, falling out of ganglion cells, increase in the glia cells and proliferation of the vessels. The greater part of the lower two thirds of the premotor area was implicated. There was, in addition, occlusion of the left posterior cerebral artery, with involvement of the structures supplied by this vessel.

CASE 5.—*Incomplete thrombosis of the left middle cerebral artery. Flaccid hemiplegia of seven months' duration. Destruction of the lower two thirds of the premotor area.*

History.—S. A., a woman, aged 48, was admitted to the Montefiore Hospital on June 15, 1932, with a history of hypertension for six years and a cerebral episode on Dec. 9, 1931, after which right flaccid hemiplegia with aphasia persisted.

Neurologic examination revealed severe motor aphasia, right flaccid hemiplegia, moderate right hyperreflexia, right homonymous hemianopia and right sensory impairment. The patient died on July 20, 1932, following bacteremia secondary to a subphrenic abscess. The flaccid hemiplegia lasted seven months.

Autopsy.—There was incomplete occlusion of the left middle cerebral artery, with destruction of the white matter of the second and third frontal and precentral convolutions as well as the left putamen and anterior limb of the internal capsule (fig. 6). Microscopic examination disclosed the same changes in the gray matter as in case 4. The lower two thirds of the premotor area was destroyed.

CASE 6.—*Hemorrhage of the lenticulostriate artery. Flaccid hemiplegia of two months' duration. Partial destruction of the white fibers of about one half of the premotor area.*

History.—Y. S., a woman, aged 52, was admitted to the Montefiore Hospital on Aug. 5, 1932, with a history of hypertension of eight years' duration and a sudden acute cerebral accident with right hemiplegia and aphasia in June 1932.

Fig. 6 (case 5).—Incomplete thrombosis of the left middle cerebral artery, causing destruction of the fibers from the second frontal and precentral convolutions.

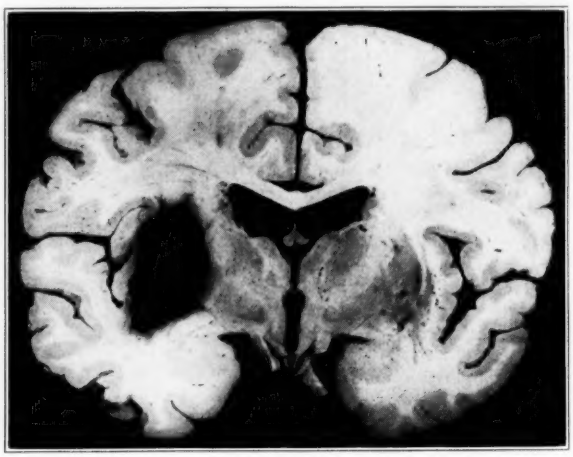

Fig 7 (case 6).—Hemorrhage of the left lenticulostriate artery. The fibers from the second frontal and precentral convolutions were destroyed.

Neurologic examination disclosed mixed aphasia of moderate degree, trophedema of the right hand, complete right flaccid hemiplegia, with right hyperreflexia, positive Babinski and Chaddock signs and right hypesthesia. The patient died suddenly on August 21. The flaccid hemiplegia lasted two months.

Autopsy.—There was a hemorrhage along the distribution of the left lenticulostriate artery, which had destroyed part of the fibers from the second and third frontal, premotor and insular convolutions, as well as the external capsule, putamen and outer segment of the left globus pallidus (fig. 7). About one half of the white fibers of the premotor area were destroyed. Microscopic examination revealed changes in the gray matter of the premotor area analogous to those in cases 4 and 5.

Comment.—This case presented flaccid hemiplegia with destruction of only about one half of the white fibers of the premotor area.

COMMENT

Analysis of the six cases of flaccid hemiplegia revealed destruction of the lower two thirds of the premotor area in the first five cases. In case 6 only half of the premotor area was affected. In the first three cases, with complete occlusion of the middle cerebral artery, the degree of involvement of the premotor area was grossly identical with that observed in cases of hemiplegia with complete closure of the middle cerebral artery accompanied by spasticity. In cases 4, 5 and 6, with incomplete occlusion of the middle cerebral arteries, the premotor cortex was not so extensively destroyed as in the first three cases. In these cases the gray matter of the lower two thirds of the premotor area on gross examination appeared partly spared. Careful microscopic examination, however, revealed histopathologic changes. These consisted of a slight distortion of the cyto-architectural layers, small areas of destruction with a dropping out of the ganglion cells, proliferation of the vessels and an increase in the glial elements.

In order to supplement our studies of involvement of the premotor area and the rôle it may play in spasticity, it was found advisable to review cases of closure of the anterior cerebral artery. As already mentioned, this vessel supplies the superior and mesial parts of the premotor area. Obstruction of this vessel therefore destroys part of the first frontal convolution and the anterior part of the paracentral lobule, which enter into the formation of the premotor area. With one exception, all of the cases of disease of the anterior cerebral artery collected by Critchley [3] presented spastic hemiplegia. The exception, Lhermitte's case, was not followed sufficiently long to exclude postictus flaccidity. Davison, Goodhart and Needles [4] also reported two cases of spastic

3. Critchley, M.: The Anterior Cerebral Artery and Its Syndromes, Brain **53**:120, 1930.

4. Davison, C.; Goodhart, S. P., and Needles, W.: Cerebral Localization in Cerebrovascular Disease, Arch. Neurol. & Psychiat. **30**:749 (Oct.) 1933.

hemiplegia caused by occlusion of the anterior cerebral artery. Thus in the cases of cerebral spastic hemiplegia the premotor area was involved.

The only cases observed by us in which the cortical premotor area was spared were four cases of flaccid hemiplegia associated with thalamic and other deep-seated lesions. Such deep-seated lesions associated with flaccidity have been recorded in the literature.

In the face of such evidence it becomes difficult to determine the relation of the premotor area to spasticity in man. Two factors favor the view that they are causally related: (1) Involvement of the premotor area existed in all of our cases of spastic hemiplegia; (2) in the only instances in which the premotor cortex was spared, flaccid hemiplegia with thalamic and other deep-seated lesions was present. Against such a thesis, however, is the absence of spasticity in the six cases of flaccid hemiplegia in which the premotor area was implicated.

A detailed discussion of the mechanism responsible for flaccid hemiplegias is not necessary, but the question of sensory disturbances in these cases must be considered. Clinicians have often emphasized this association. An analysis of clinicopathologic observations, however, militates against the view that these sensory disturbances play a rôle in the production of flaccidity. Sensory disturbances were elicited in all of our cases of flaccid hemiplegia. Similar sensory changes, however, were observed in all of the cases of complete thrombosis of the middle cerebral artery resulting in spasticity. Experimentally, decortication in primates also produces spasticity despite the removal of the postcentral gyri.

SUMMARY

In view of the production of spasticity by ablation of the premotor area in primates and by the removal of a glioma (Kennard, Viets and Fulton [2]) in the premotor region which induced spasticity in a man, a correlation between disease of the premotor area and spasticity in man was attempted. Two groups of cases of hemiplegias, spastic and flaccid, were studied. Involvement of the premotor area was present in all of the cases of spastic hemiplegia. In six cases of flaccid hemiplegia, the premotor area was also implicated. In the first three of these six cases the degree of destruction was as marked as in any of the cases of spastic hemiplegia due to complete obstruction of the middle cerebral artery. The only cases of flaccidity in which the premotor cortex was spared were four cases of thalamic and deep-seated lesions.

CONCLUSIONS

Clinicopathologic investigations of this series reveal: (1) that the premotor area in man may be involved without giving rise to spasticity, and (2) that the integrity of the premotor area is not solely responsible for the presence of flaccidity.

TUMORS OF RATHKE'S CLEFT

(HITHERTO CALLED TUMORS OF RATHKE'S POUCH)

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AND

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PHILADELPHIA

The nomenclature of pituitary and parapituitary lesions needs revision. In the first place, the term pituitary should be accepted and that of hypophysis discarded, or *vice versa*. Since the term pituitary, derived from the root *pituita,* or phlegm, was adopted by the ancients because the structure was believed to supply the mucous secretion to the nasal membrane, we suggest abandoning it and accepting the alternate term hypophysis.

There is general acceptance of the terms applied to the component parts of the hypophysis, namely, the pars anterior, pars intermedia and pars posterior, although Tilney proposed that the pars anterior be called the pituitary gland and the pars posterior (the neural lobe) the processus infundibuli.

However, this communication does not concern the hypophysis itself but the parahypophyseal lesions, which are commonly called tumors of Rathke's pouch and craniopharyngeal tumors or craniopharyngiomas. But these last two terms, as Tilney says,[1] are unquestionably misnomers and misleading. In its origin a tumor of Rathke's pouch has no relation to the pharynx. The so-called craniopharyngioma, on the contrary, takes its origin from an anlage of the original stalk from the buccal cavity. As a substitute for craniopharyngioma we suggest the term tumor of *the hypophyseal stalk*. Tilney proposed the term bucconeural tumor. Throughout this communication we shall refer to these tumors as "tumors of the stalk."

Finally, the term tumor of Rathke's pouch, for obvious reasons, as we shall show later, should give way to tumor of Rathke's cleft. In this brief communication we shall endeavor to define a tumor of Rathke's cleft anatomically and pathologically, so that it can be identified and acknowledged as a definite pathologic entity and can be differentiated from other hypophyseal or parahypophyseal lesions.

Read before the Philadelphia Neurological Society, April 27, 1934.

From the Clinic and Laboratory of Neurosurgery in the Hospital of the University of Pennsylvania.

1. Personal communication from Dr. Frederick Tilney, dated March 15, 1934.

In the terms of the anatomist, Rathke's pouch is an invagination from the roof of the primitive oral cavity. In the process of development it becomes constricted off, and a sac with a narrow stalk is formed. The sac eventually closes, and the stalk, originally connected with the mouth, usually disappears. This stalk in some cases persists, as does the thyroglossal duct (the analogy is close), and it is from these epithelial rests that tumors of the stalk have their origin.

The original Rathke's pouch becomes converted into a solid structure, the pars anterior or the glandular lobe of the hypophysis. But *a small cleft* remains between the pars anterior and the pars posterior or

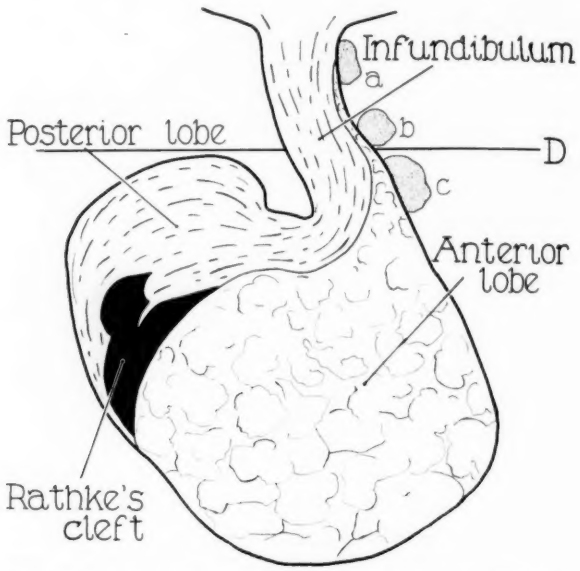

Fig. 1.—Anatomic drawing to show the various constituents of the fully developed hypophysis. Note the location of Rathke's cleft between the anterior lobe and the posterior lobe. The situations of the epithelial rests, which give rise to tumors of the stalk, are indicated by *a*, *b* and *c*. *D* indicates the diaphragma sellae.

processus infundibuli. This is all that remains of the original pouch, as described by Rathke, and it is from this cleft that the tumors under discussion originate. In this sense it seems proper to designate them as tumors of Rathke's cleft (fig. 1).

In the clinic, when one is confronted with tumors related anatomically or embryologically to the hypophysis, one must differentiate between an adenoma, tumor of Rathke's cleft and a tumor of the stalk. Each of these three lesions, because of its proximity to the hypophysis, may be accompanied by signs of glandular dysfunction. As a rule,

however, an adenoma may be differentiated from a tumor of Rathke's cleft and a tumor of the stalk (1) by the characteristic glandular picture (it may be either of the two common types, chromophil or chromophobe), (2) by the distortion of the field and (3) by the unmistakable deformation of the sella turcica. On the other hand, a tumor of the stalk can usually be differentiated from an adenoma by the fact that a tumor of the stalk develops usually in children, whereas the adenoma develops in the adult; a tumor of the stalk is frequently cystic, and there are often calcified deposits in the tumor or in the wall of the cyst. A tumor of the stalk is chiefly a suprasellar lesion, whereas an adenoma is intrasellar. Because of its proximity to the third ventricle tumor of the stalk often establishes a ventricular block, with the consequent papilledema and secondary postpapillitic atrophy. On the other hand, primary optic atrophy is a signature of the adenoma.

Thus, from the clinical point of view there is no difficulty in differentiating a tumor of the stalk from an adenoma. It must be remembered,

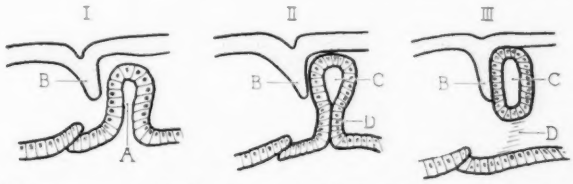

Fig. 2.—Diagrammatic sketch of the development of the hypophysis from the buccal cavity modified from Bailey (Intracranial Tumors, Springfield, Ill., Charles C. Thomas, Publisher, 1933, p. 96): *I*, the original invagination from the buccal cavity (*A*); *II*, the communication with the buccal cavity, obliterated at *D* and the structure from which the anterior lobe (*C*) arises, and *III*, a remnant of the stalk (*D*), showing the anterior lobe (*C*) in close relation with the infundibular process (*B*).

however, that there are two recognized locations for a tumor of the stalk, a lower and a higher, according to whether the tumor takes its origin from the lower or the higher groups of epithelial anlagen in the duct (fig. 2).

So far as our experience goes, it is impossible by any method of examination to say with certainty before operation that a lesion is a tumor of Rathke's cleft. A tumor may grow within the sella turcica and may simulate an adenoma in its sellar deformation (fig. 4) and in the signs of glandular dysfunction, or it may enlarge upward beyond the confines of the sella turcica and may reproduce the signs of a low-lying tumor of a duct without calcification.

In conversation with roentgenologists, in reviewing their reports and in conversation with neurologists, one of us (C. H. F.) has found that the terms tumor of Rathke's cleft and tumor of the stalk have been

used interchangeably. This confusion as to terms has existed for a number of years. But it must be remembered (1) that a tumor of the stalk does not arise from Rathke's pouch, and (2) that a tumor of Rathke's cleft, as we shall demonstrate, differs histologically not only from a tumor of the stalk but from all other lesions in the neighborhood of the sella turcica. We shall attempt to establish the tumor of Rathke's cleft as a definite entity.

REPORT OF A CASE

For purposes of illustration we present the following case:

Frontal headaches and ocular trouble for two years. Blindness of the right eye for four months. Temporal hemianopia in the left eye. Blurring of the disks. Erosion of the right sphenoidal ridge. Transfrontal craniotomy with disclosure of the cyst presenting between optic nerves. Histologically a true cyst of Rathke's cleft with a columnar ciliated epithelial lining.

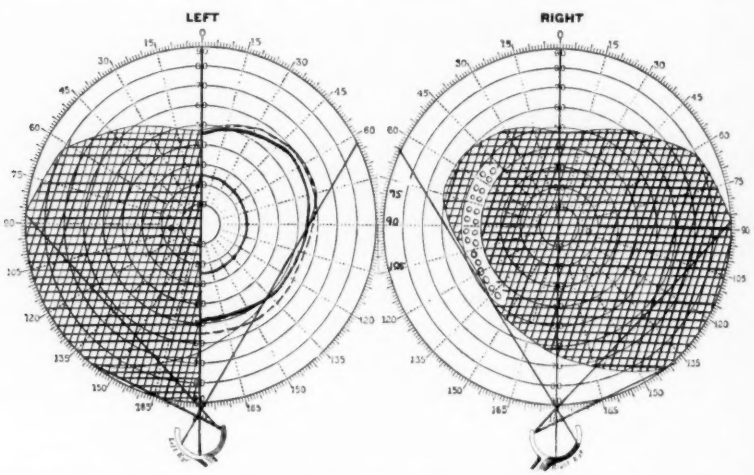

Fig. 3.—Preoperative fields (July 28, 1931). Vision was: left eye, 3/60; right eye, shadows.

History.—C. B., a man, aged 52, was referred to the neurosurgical service of the Hospital of the University of Pennsylvania on July 27, 1931, by Dr. R. D. Wallace. About two years before entrance he had begun to have frontal headaches. He was treated for sinus disease, and the headaches disappeared after eleven weeks. They returned eight months before entrance and persisted from this time on. They were dull, constant and mostly frontal, although for two months before entrance into the hospital the patient had pain in the left temple.

In 1929, about two years before admission, he began to have trouble with his eyes. Objects were at first blurred and indistinct. Vision became steadily worse, until it was completely lost in the right eye four months before the present writing.

Laboratory Examination.—There were blindness of the right eye, temporal hemianopia in the left eye (fig. 3) and yellow optic disks which were blurred along the margins. There were no other positive findings. The spinal fluid pressure was 150 mm. of water. Serologic tests of the blood and spinal fluid gave negative results.

A roentgenogram of the skull showed slight erosion of the right sphenoid ridge. The pituitary fossa measured 11 by 8 mm. and showed no erosion or enlargement.

Operation.—On July 29, 1931, a right transfrontal craniotomy was done under avertin anesthesia. The usual dual flaps (the scalp flap and the bone flap) were reflected separately, as for pituitary exploration. It was apparent that there was

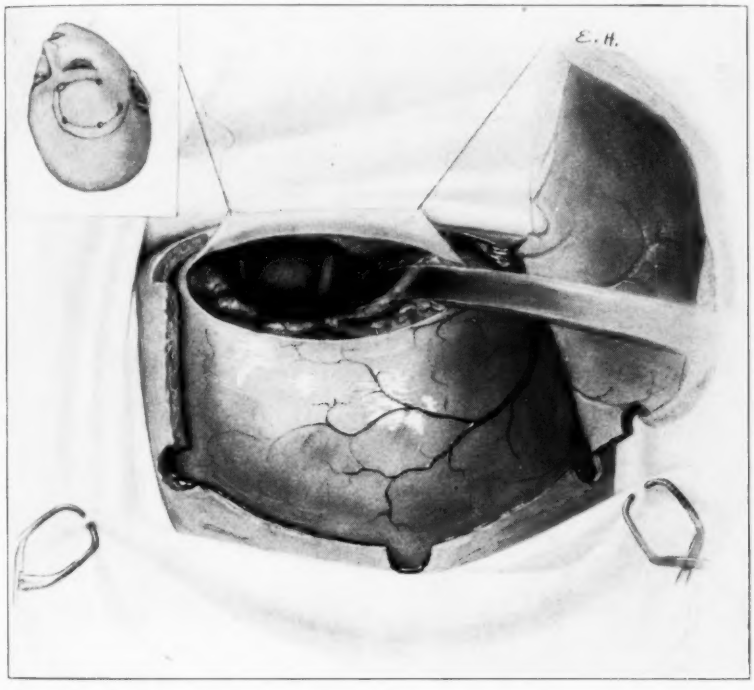

Fig. 4.—Drawing showing the approach to the sella turcica from the right side, following the direction of the greater wing of the sphenoid bone. The tumor and its relation to the optic nerves are evident.

no increased intracranial pressure, and as the frontal lobe was elevated, an unusual amount of cerebrospinal fluid was evacuated. This so reduced the pressure that a clear view could be had of the entire anterior fossa from right to left. There was bluish membrane between the optic nerves, evidently the wall of a cyst (fig. 4). This was readily freed from the right and left optic nerves, and 8 cc. of a brownish fluid was evacuated from the cavity of the cyst. The wall of the cyst collapsed, leaving a considerable space between it and the optic nerves. The chiasm was displaced backward, and the distance from the optic foramens to the chiasm was at least 3 cm.

The wall of the cyst was incised, and the interior minutely inspected. There was not a vestige of solid tissue. A considerable portion of the wall was

removed (fig. 5), and what remainead was swabbed with a 5 per cent solution of tincture of iodine. The flap was replaced, the wound closed and drainage established through a stab wound.

Postoperative Course.—The patient's convalescence was uneventful, and he was discharged from the hospital on August 13 (fig. 6). He remained free

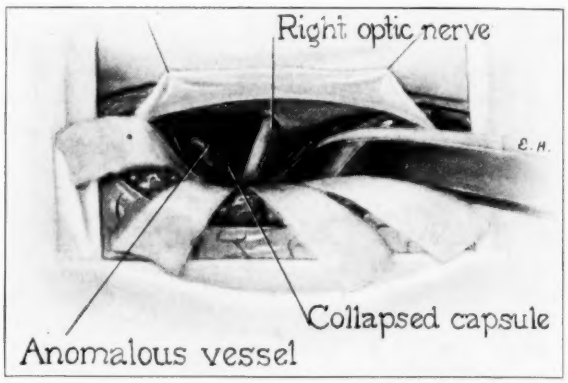

Fig. 5.—The collapsed capsule after the evacuation of the cyst.

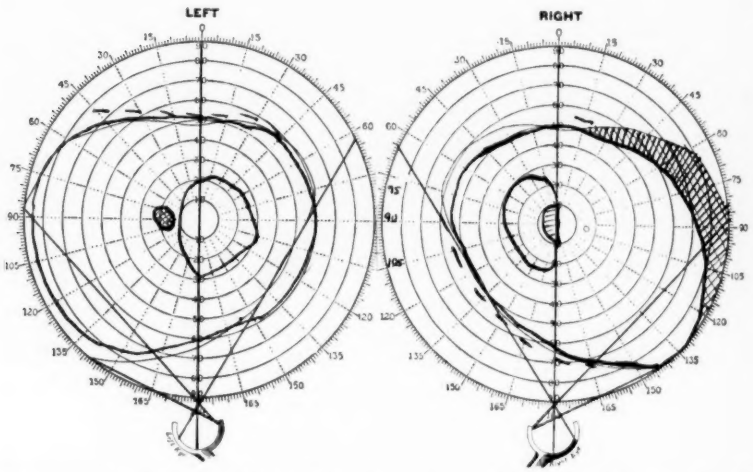

Fig. 6.—Postoperative fields (Feb. 10, 1932). Vision of the left eye was 6/12; of the right eye, 6/60.

from headaches after the operation, gained weight and was able to return to work. He was well when last seen, almost three years after the operation.

Pathologic Report.—Study of the wall of the cyst removed in this case revealed a type of tumor which has not been encountered in any of the other parahypophyseal lesions in the neurosurgical service. The tissue which was removed at operation was presumably characteristic of the specimen. Part of the wall of the cyst was

removed, and the exposure of the tumor at operation was clear enough so that it was possible to see the entire tumor and to determine that it was completely cystic.

Histologic study revealed a rather thin piece of tissue in which there were many necrotic areas as well as large fibrotic portions. In other parts there were large collections of cells with a rather foamy appearance. They had small, round nuclei with many coarse clumps of chromatin and abundant foamy cytoplasm and looked very much like xanthomatous cells. Differential stains for acidophilic and basophilic granules revealed no evidence of a differentiated granular structure. Mucin stains revealed no evidence of mucin in the ciliated cells. The nature of the cells could not be determined accurately, but they appeared to be very much swollen and distended and possibly of pituitary origin.

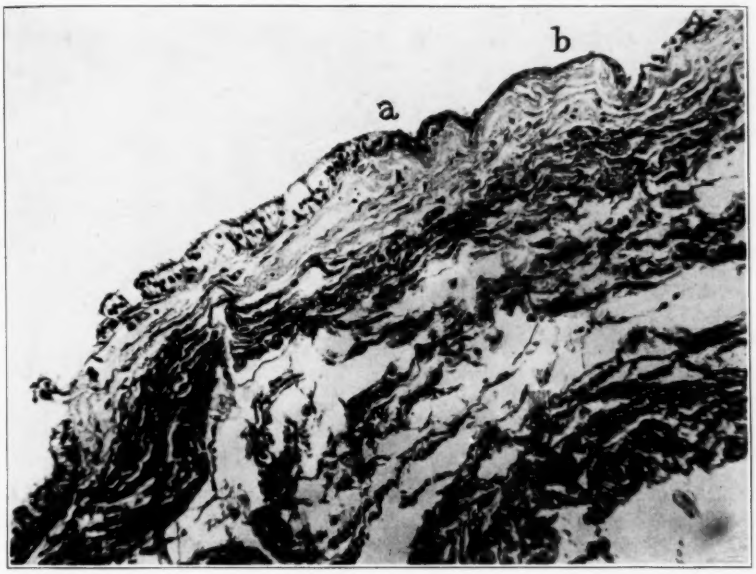

Fig. 7.—A low power photomicrograph showing the lining of the ciliated columnar cells which became cuboidal (*a*) and finally flat (*b*). Hematoxylin and eosin stain.

There were large portions of the wall of the cyst which were completely fibrotic. Blood vessels were numerous, especially under the outer layer. They consisted chiefly of large cavernous spaces and of small capillaries.

The feature of outstanding interest, however, was the presence of a single layer of ciliated columnar epithelium lining the cyst. This layer of cells was continuous around the wall. Most of the cells were columnar, but there were many transitions from these columnar cells to cuboidal and even flat cells (fig. 7). Occasionally there seemed to be a tendency toward the formation of more than a single layer of cells. The columnar cells contained round nuclei with stout nuclear membranes and a coarse chromatin structure, which filled the cell. Usually they were situated in the bottom of the cell, but they were sometimes visible in the center of the cell and even close to the top. The cytoplasm had a most

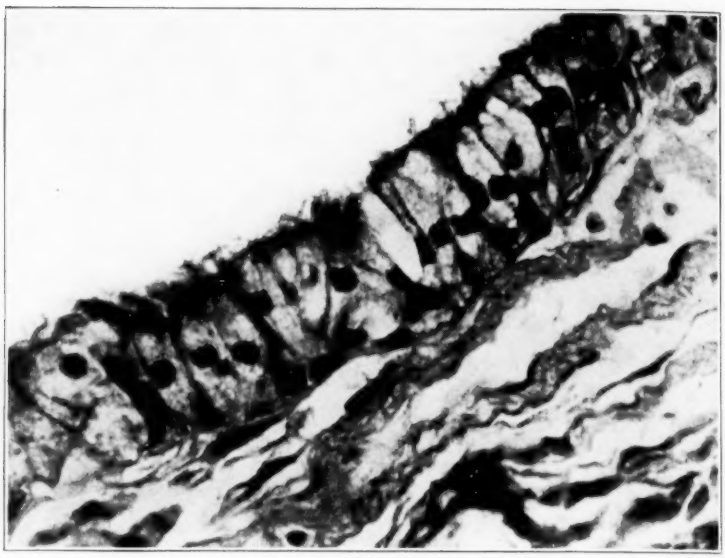

Fig. 8.—A high power photomicrograph showing the ciliated cells with their vacuolated cytoplasm and their stout cilia. Hematoxylin and eosin stain.

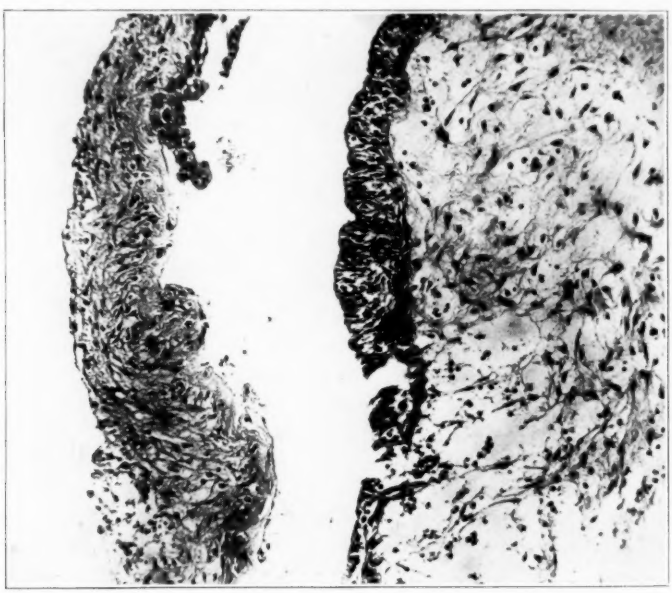

Fig. 9.—A small portion of the tumor with an area of squamous epithelium which is not directly connected with the ciliated part.

peculiar appearance; it was foamy, granular and distended. It appeared to be very much like the cytoplasm in the goblet cells of the stomach. Further differential granular stains revealed an incapacity on the part of the cytoplasm to take an acidophilic or a basophilic stain.

Stout cilia projected from the tops of the cells in large numbers (fig. 8). Basal bodies were readily demonstrated. While this ciliated columnar epithelium was not present along the entire wall of the cyst, transitions from it to cuboidal or flat cells could be traced. The cuboidal cells contained cilia, while the flat cells did not.

In one portion of the tissue there was a small area which was composed of stratified squamous epithelium (fig. 9). This tissue had no direct connection with the ciliated cells lining the cyst. Some of the superficial cells in this area seemed to be ciliated, but the bodies were not clear (fig. 10).

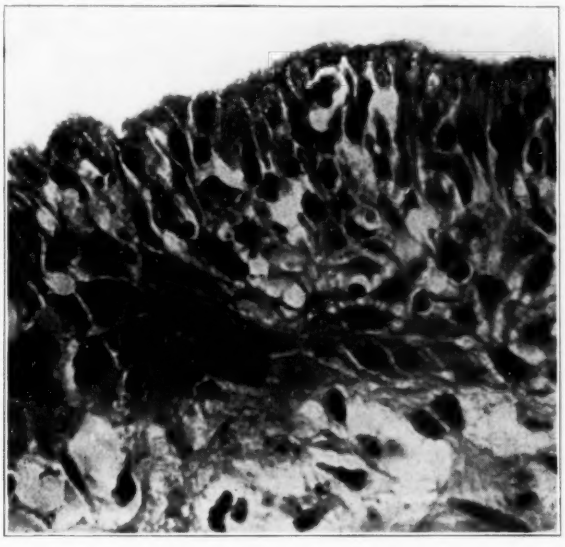

Fig. 10.—The area of squamous epithelium under high magnification, showing clearly the stratified nonciliated structure.

COMMENT

The tumor reported here was situated entirely in the suprasellar area, with no visible connection with the third ventricle. Its structure and, in part, its location are suggestive of a congenital tumor originating from the remnants of Rathke's cleft. For a long time we have thought that the term cyst or tumor of Rathke's pouch was used too loosely, and that most of the tumors so designated were really of a different type. We know now that the majority of so-called tumors of Rathke's cleft are in reality tumors derived from the hypophyseal stalk, and that few if any really originate in Rathke's cleft. The separation of this type of tumor from others of the group occurring in and about

the sellar region is desirable in order better to define histologically the various lesions in this region.

That there is histologic justification for the separation of this tumor from other congenital tumors in the sellar region is demonstrated by the studies of Duffy,[2] who found that the hypophyseal vesicle or sac, a later stage of Rathke's pouch, is composed of stratified cylindric epithelium. Most of this tissue develops into the anterior lobe, but a single layer of cylindric epithelium persists in the adult gland as the "cleft" (Rathke's cleft), separating the anterior and posterior lobes. On the contrary, "the hypophyseal duct is composed of modified squamous epithelium which gradually passes over into the cubical epithelium of the buccal canal." Duffy believed, furthermore, that the cysts of Rathke's cleft should be differentiated from those of squamous epithelial origin. He found that cysts of Rathke's cleft were lined with cylindric epithelium, often ciliated. He stated that

> . . . in general, one may say that unless there is strong reason to suspect an ependymal or colloidal glandular (i. e., from the acini of the pars anterior) origin for such cysts lined with a single layer of ciliated columnar epithelium, they may properly be attributed to an origin from the cleft or from a diverticulum of the pouch of Rathke.

The feature in our case which differentiates the tumor from other tumors in the region of the sella turcica is the presence of a single layer of ciliated columnar epithelium. This is suggestive of an origin from the cleft of Rathke, though similar cells may arise from the ependymal lining. The lack of connection with a ventricular cavity and the presence of columnar rather than cuboidal cells would not indicate an ependymal origin. Embryologic studies, so far as we are aware, have not demonstrated ciliated cells in Rathke's cleft. The studies of Duffy appear to be alone in their demonstration of such cells. Rathke's cleft being derived from the buccal cavity, is composed of stratified epithelium of a nonciliated character. Whether ciliated cells may be included in the entodermal portion of the hypophysis or the so-called pouch of Seessel is open to grave doubt, inasmuch as the participation of the entoderm in hypophyseal development is doubtful. While some investigators (Smith[3] and Miller[4]) stated the belief that a portion of the anterior lobe of the hypophysis in ganoids and pig embryos is derived from the entoderm, the work of Atwell[5] failed to substantiate it. In

2. Duffy, W. C.: Hypophyseal Duct Tumors, Ann. Surg. **72**:537 and 725, 1920.

3. Smith, P. E.: The Development of the Hypophysis of Amia Alva, Anat. Rec. **8**:499, 1914.

4. Miller, M. M.: A Study of the Hypophysis of the Pig, Anat. Rec. **10**: 226, 1916.

5. Atwell, W. J.: The Development of the Hypophysis Cerebri of the Rabbit, Am. J. Anat. **24**:271, 1918.

his study of the development of the human hypophysis, Atwell stated unequivocally, "I have been unable to satisfy myself that Seessel's pouch becomes incorporated with or even makes a considerable contribution to the developing human hypophysis." The evidence, therefore, for the participation of the entoderm in the development of the human hypophysis is too meager to permit conclusions concerning the origin of ciliated cells from this portion of the embryo.

The possibility of the origin of these ciliated cells and cysts from the pars tuberalis of the hypophysis suggested itself, but so far as is known, this portion of the hypophysis contains no ciliated cells. Nevertheless, the fact remains that there are in the human hypophysis ciliated cells which are found in the so-called Rathke cleft. Bryant [6] claimed that ciliated cells were to be found in all freshly hardened human hypophyses, but this has lacked confirmation by others. The incidence of such cells is apparently not great. Rasmussen [7] found ciliated epithelium in only two of three hundred hypophyses. Kiyono [8] found it in one of fifty cases and Guizzetti [9] in three of fifty-four cases. When ciliated cells are present, they are found in the cyst of Rathke or the so-called pars intermedia. In Rasmussen's cases they were found on the side of the residual lumen next to the anterior lobe; only a few were on the opposite wall. They are columnar, with a sharply defined free border, beneath which are the basal bodies, and with more or less granular or vacuolated cytoplasm. The presence of ciliated epithelium lining the Rathke's cysts was described by Erdheim [10] in man and by Martin [11] in rats and marsupials. The occurrence of the lining was conceded by Bailey,[12] who stated that the cystic cavities between the anterior and posterior lobes of the hypophysis in man are lined with ciliated epithelium. The significance of these ciliated cells is uncertain. Rasmussen suggested that they may be pathologic or that "they should be looked upon as unusual differentiations of hypophyseal tissues or

6. Bryant, W. S.: Sensory Elements in the Human Cerebral Hypophysis, Anat. Rec. **11**:25, 1916.

7. Rasmussen, A. T.: Ciliated Epithelium and Mucin Secreting Cells in the Human Hypophysis, Anat. Rec. **41**:273, 1928.

8. Kiyono, H.: Ueber das Vorkommen von Plattenepithelherden in der Hypophyse, Virchows Arch. f. path. Anat. **252**:118, 1924.

9. Guizzetti, P.: Sulla struttura della pars intermedia dell'hypophysis cerebri dell'uomo, Sperimentale, Arch. di biol. **80**:665, 1926.

10. Erdheim, J.: Pathologie der Hypophysengangsgeschwülste, Ergebn. d. allg. Path. u. path. Anat. **21**:482, 1926.

11. Martin, T.: Ciliated Cells in the Anterior Lobe of the Hypophysis, Compt. rend. Soc. de biol. **113**:216, 1933.

12. Bailey, P.: The Structure of the Hypophysis Cerebri of Man and of the Common Laboratory Animals, in Cowdry, E. V.: Special Cytology, New York, Paul B. Hoeber, Inc., 1928, vol. 1, p. 489.

as migrations of nasopharyngeal elements during early stages of development."

Whatever the origin of the ciliated cells may be, it is certain that at least in some human hypophyses such cells are found lining the remains of Rathke's cyst, and that they conform in their morphology to the type of cell seen in our case. Theoretically, tumors of Rathke's cleft should be found within the sella turcica, but when one considers the origin of the anterior lobe from the buccal epithelium and the process of traversal and rotation during the course of its development, it is not surprising to find such tumors wholly outside the sella turcica.

The question arises whether a tumor such as we describe could be derived from tissues other than those of the hypophysis. The tumors may be ependymal in origin, but for reasons previously stated this is not likely. Cysts similar in all respects to the one in the case which we have reported have been described as colloid tumors of the third ventricle, but it is difficult to connect them with cysts of Rathke's cleft. A tumor of this sort, however, was recorded by Greenfield, who suggested that it may have been of hypophyseal origin. Most of the cysts in the third ventricle lined by ciliated epithelium probably originate in the ependyma or choroid plexus. Moreover, while the wall lining such a cyst is similar to that of the cyst of Rathke's cleft in our case, the tumors are otherwise dissimilar.

CONCLUSIONS

1. We propose the following revisions in the nomenclature of these sellar and parasellar lesions: (*a*) the term hypophysis should be retained and pituitary discarded; (*b*) as it is a misnomer and misleading, the term craniopharyngioma should be discarded in favor of tumor of the hypophyseal stalk, and (*c*) so-called tumor of Rathke's pouch should be designated tumor of Rathke's cleft.

2. There are rare tumors in the region of the sella turcica lined by ciliated epithelium.

3. These tumors constitute what we designate as true tumors of Rathke's cleft.

4. They should not be confused with any ill-defined group of what are frequently called tumors of Rathke's pouch, which we believe are mostly tumors of the hypophyseal stalk.

5. These tumors have certain characteristics by which they may be differentiated from other adjacent lesions.

CHILDREN'S IMAGINARY COMPANIONS

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The phenomenon of imaginary companions, which is usually regarded as normal in children, but which when encountered in adults suggests a psychopathologic condition, appears to have had little investigation. However, numerous theories in regard to its significance have been advanced, some of which are sociologic, others psychologic and others psychoanalytic.

SIGNIFICANCE OF THE PHENOMENON

Sociologic Theories.—Cooley [1] observed that:

> Some children appear to live in personal imagination from the first month. Others occupy themselves in early infancy mostly with solitary experiments upon blocks, cards and other impersonal objects, and their thoughts are doubtlessly filled with images of these. . . . People differ in the vividness of their imaginative sociability. . . . There is no separation between real and imaginary persons; indeed to be imagined is to become real in a social sense.

Mead [2] reported that in New Guinea there are for the children no socially defined lacks in their lives and consequently no imaginative structures.

> It is not until the Manus boys reach their teens that any need is felt. But after losing their fathers, they do feel definitely bereaved, socially maimed, so to speak, and it is at this age that the only imaginative play takes place.

Psychologic Theories.—Faegre and Anderson [3] stated:

> Like primitive man, the child attributes to natural objects the same thoughts and feelings that move him. If we respect his wish not to sit on the chair where his imaginary guest is sitting, we are adding to his fun.
>
> It is sometimes assumed that there is something morbid about imagination and that its appearance in the child is to be feared and repressed. . . . There is no more danger connected with the imagination than with any other natural mental ability.

From the Institute for Juvenile Research.

1. Cooley, Charles H.: Human Nature and the Social Order, New York, Charles Scribner's Sons, 1922.

2. Mead, Margaret: Growing Up in New Guinea, New York, William Morrow & Company, 1930.

3. Faegre, Marion L., and Anderson, John E.: Child Care and Training, Minneapolis, University of Minnesota Press, 1930.

Hall [4] said: "The imaginary companion shows the impulse of a lonely child to find an ideal world with sympathy and companionship."

Harvey [5] found that:

An imaginary playmate is a visual or auditory idea that becomes as vivid as a visual or auditory percept would be. The principal distinction between the idea which is projected as the imaginary playmate and other ideas is found in the unusual strength of the centrally initiated impulse which accompanies the process. Children generate a great amount of nervous energy, and, as a consequence, their centrally initiated impulses are more likely to approximate the strength of peripherally initiated impulses than are the centrally initiated impulses of grown persons.

Kirkpatrick [6] wrote:

A careful study of the matter shows that not only do a few lonely and highly imaginative children have these companions, but nearly all children have them in some form for a greater or less period of time.

These imaginary companions frequently appear in the third year when the child is getting acquainted with his own variable personality which is sometimes "nice" and sometimes naughty, or in connection with early experiences with a playmate who is not present all the time, or after hearing of a little boy or girl of a certain character.

Sometimes the imaginary companion is an ideal self, sometimes a naughty scapegrace, and at other times not self at all, but a distinct personality.

Klüver,[7] writing of an eidetic image of the B-type, to which this phenomenon appears to be related, said:

It is often nothing but a visualized idea projected into perceived space; whatever the subject is thinking about is visualized with sensory vividness. The subject can, without effort, produce EI and at pleasure banish them. He can do this without a preceding presentation of a stimulus.

Munroe [8] stated:

The imaginary companion appears to play a double rôle, that of playmate, and also a sort of self-projection or idealization of the child who created him. It represents, I think, the free will of the child aroused by the mother's admonition.

Norsworthy and Whitley [9] wrote:

Images of children tend to be more vivid, more intense than those of adults. This opinion is based on the fact that it is sometimes difficult to get little children

4. Hall, G. Stanley, and others: Aspects of Child Life and Education, edited by T. L. Smith, Boston, Ginn and Company, 1907.

5. Harvey, N. A.: Imaginary Playmates and Other Mental Phenomena of Children, Ypsilanti, Mich., State Normal College, 1919.

6. Kirkpatrick, E. A.: Fundamentals of Child Study, New York, The Macmillan Company, 1929.

7. Klüver, Heinrich: The Eidetic Child, in Murchison, Carl: Handbook of Child Psychology, Worcester, Mass., Clark University Press, 1931.

8. Munroe, J. P.: Pedagog. Sem. **3**:182 (Oct.) 1893.

9. Norsworthy, W., and Whitley, M. T.: Psychology of Childhood, New York, The Macmillan Company, 1918.

to distinguish between memory images and images of the imagination. In some children the confusion goes further. They cannot distinguish between percepts and images.

The creation of imaginary companions is a result of this confusion. The presence in a child's life of imaginary companions is very much more common than has been supposed.

Piaget [10] wrote:

The child is impelled, even when he is alone, to speak as he acts. If a child talks when he is alone as an accompaniment to his action, he can reverse the process to bring about what that action of itself is powerless to do. Hence the habit of romancing or inventing, which consists in creating reality by words and magical language alone, apart from any contact with people or things. . . . .

The child soliloquizes even in the presence of other children, although in some cases it accelerates action, it also runs the risk of supplanting it. This is the mechanism of solitary games, where after thinking his action aloud, the child under the influence of verbal excitement as much as of any voluntary illusion, comes to command both animate and inanimate beings.

The child's mind is full of ludistic tendencies up to the age of 7 or 8, which means that it is extremely difficult for him to distinguish between fabrication and truth.

Tanner [11] wrote:

Sometimes, distance and mystery are great stimulants to a child's fancy. Probably most children have fictitious characters with whom they play at times, but the imaginary playmate reaches its fullest development in the child who plays alone. . . .

It is frequently the case that the tendency to create such companions is hereditary.

Terman [12] said:

A good many gifted children have had imaginary playmates or imaginary countries, but comparative data on this point are not available for unselected children.

There is some indication that they occur more frequently in cases where the child has no real playmate.

Vostrovsky [13] wrote:

Most of us do not realize how common it [the phenomenon of imaginary companions] is, and how large a part it can play for evil as well as good in a child's life. That companions are not the result of defective organization is shown in their being found in active healthy children when thrown much alone.

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10. Piaget, Jean: The Language and Thought of the Child, New York, Harcourt Brace and Company, Inc., 1926.

11. Tanner, Amy Eliza: The Child: His Thinking, Feeling and Doing, Chicago, Rand-McNally & Company, 1904.

12. Terman, Lewis M.: Genetic Studies of Genius, Palo Alto, Calif., Stanford University Press, 1926, vol. 1.

13. Vostrovsky, Clara: A Study of Imaginary Companions, Education **15**:383, 1895.

Psychoanalytic Theories.—Green [14] found:

> The daydream permits expression of instinctive motives which are repressed in the real world. . . . The imaginary companion indicates a desire for self assertion and display.

Von Hug-Hellmuth [15] said:

> When a child assumes a new rôle in phantasy, it is usually of the same sex as himself. He wishes to represent himself as occupying positions of no less importance than those held by persons who have some real or imaginary advantages over him.
>
> In this playing the child is able to live out his hostile impulses toward his parents, brothers and sisters, without being obliged to fear punishment, as he can stop his play at any moment. . . .
>
> The value of playing with words is found particularly among those intelligent children upon whom early education has imposed restrictions for which the little people do not understand the reasons.

Wickes [16] stated:

> The imaginary playmate is used to integrate warring elements, eliminate the undesirable, furnish a pattern, compensate for a failure and as an excuse for retreat.

THE PROBLEM AND METHOD

The present study was undertaken with a view to focusing attention on some of the individual and environmental factors thought to be associated with the phenomenon. Psychologic tests and technics of social investigation were used to supplement each other, and comparisons were made with a control group.

The term imaginary companion, as it is used in this study, implies an invisible character, named and referred to in conversation with other persons or played with directly for a period of time, at least several months, having an air of reality for the child, but no apparent objective basis. This excludes that type of imaginative play in which an object is personified, or in which the child himself assumes the rôle of some person in his environment.

To illustrate: Shortly before her second birthday Mary referred to "Tagar," her first imaginary companion. She led Tagar around on an imaginary string. Food was kept for it under the radiator where it also slept; she always fed it on the floor. It was particularly fond of ice cream, as she was. "Berrie and Auntie" followed Tagar, appearing when she was about 3½ years of age. They were two persons, but lived together. Mary would set places for them at the family table. Although dishes and silver were laid and Mary would ask if they had had enough, real food was never offered them. On other occasions she would seize the oppor-

14. Green, G. H.: Psychoanalysis in the Classroom, New York, G. P. Putnam's Sons, 1922.

15. von Hug-Hellmuth, H.: Study of the Mental Life of the Child (translated from the German by James T. Putnam and Mabel Stevens), Monograph 29, Washington, D. C., Nervous and Mental Disease Publishing Company, 1919.

16. Wickes, Frances G.: The Inner World of Childhood: A Study in Analytical Psychology, New York, D. Appleton and Company, 1927.

tunity at meal-time to tell her father all the things which they had done. Mary might be punished but they never were, and never did anything wrong. Berrie and Auntie frequently accompanied her and her parents on outings, and on several occasions she attempted to draw her real companion into play with them, by insisting that she talk with them on the telephone.

Specific questions for investigation in this study were:

1. Is this experience common to all children, is it peculiar to certain types of children or is it related to given environmental conditions?

2. To what chronological age or developmental period is it peculiar?

3. Is there any correlation of this phenomenon with general intelligence, as measured by tests?

4. Can anything be learned of the sources of the fantasy material?

5. How is the phenomenon regarded and handled by parents?

Selection and Investigation of Cases.—Permission was received to appear at the regular monthly meetings of the mothers' group of a Chicago suburb, which includes families of all social and economic levels. A brief explanation of the proposed study was followed by a request for cooperation in the discovery of cases. Data were secured on 40 children, each of whom had had one or more imaginary companions for a minimum period of several months. In the presentation of the study, mention was made of the theory that imaginary companions are thought to be a phenomenon characteristic of intelligent children, to allay any fear that it might be regarded as abnormal.

The children in the group thus selected ranged from 3 to 16 years of age. Information secured directly from the child in a recorded interview was supplemented by a social history furnished by the mother in a personal interview, a school report of academic and social adjustment and an intelligence test. Forty kindergarten children in the same community served as a control group.

OBSERVATIONS AND RESULTS

Incidence.—Vivid and sustained imaginary companions are not common to all children. The incidence among 119 children more than 4 years of age in forty-six unselected families in this community was 13.4 per cent. Other investigators have reported somewhat different findings. Harvey,[5] while reporting an incidence of 6 per cent, suggested that it may be greater. Hurlock and Burnstein [17] reported 31 per cent among girls and 23 per cent among boys. The discrepancy of these figures with the findings of the present study may be partially accounted for by the fact that Hurlock and Burnstein included less sustained experiences.

It is interesting to observe that whereas in some families the phenomenon is never encountered, four instances were reported for 2 children in the same family. In each of these families the siblings were

17. Hurlock, E. B., and Burnstein, M.: The Imaginary Playmate: A Questionnaire Study, J. Genet. Psych. **41**:380 (Dec.) 1932.

widely separated in chronological years, so that presumably it was not a case of imitation.

The observations of Vostrovsky [13] and of Hurlock and Burnstein [17] that the phenomenon is encountered more often in girls, is substantiated by this study. The distribution between the sexes is: female, 75 per cent, male, 25 per cent.

Individual Factors.—1. Intelligence: Considering the small number of cases studied, the age range of the children, the fact that four different individual tests [18] were utilized and possible selective factors in the community from which the group was chosen, perhaps no attempt should be made to discuss intelligence; yet it seems important, since there is current the impression that imaginary companions are peculiar to children of superior intelligence. The findings of intelligence tests, administered by staff psychologists of the Institute for Juvenile Research, are presented, since, in general, the results of the four tests correlate closely.

The median intelligence quotient for the 40 selected children was 120, yet 2 children in the group had intelligence quotients in the nineties, indicating that the phenomenon is not limited to children with superior intelligence, though probably more prevalent among them. One case in a markedly defective child has come to my attention. A study of sixth grade children by means of the Otis group test showed the mean intelligence quotient of the sixth grade school population in this community to be 107. The selected group was therefore above the average of the community in intelligence. On the assumption that tests of school children are likely to be more dependable than those of preschool children, the median intelligence quotient for the selected children above 6 years of age was computed and found to be 116, a figure still above the average. The impression that children with imaginary companions tend to be above the average in intelligence was further substantiated by the fact that among the unselected kindergarten children the 6 children who were found to have had this experience were all in the upper third of their class so far as intelligence goes.

2. Physical Status: Medical histories secured from mothers reveal little that is significant. Among the 40 selected cases there were 2 instances of physical abnormality, one a harelip, and the other flatfoot. There were also 2 cases of stuttering and 2 of left-handedness. Possible constitutional differences call for a much more refined analysis.

3. Personality: Writing in 1895, Vostrovsky [13] associated the phenomenon with a nervous temperament. Psychologic investigators since that time have given scant consideration to the behavior mechanisms of the child himself, although they have described the phenomenon and called attention to possible environmental conditioning factors.

In the absence of adequate personality tests for children, descriptive data in regard to personality were secured from the adults in the child's environment.

18. The intelligence tests used were the Stanford-Binet, the Kuhlman Binet, the Merill-Palmer and the Army Alpha.

Personality difficulties in some form were reported by mothers for 35 of the 40 selected children. The more common of these in the order of frequency were: timidity in the presence of other children; a domineering manner with other children; fear in physical activity; sensitivity; an undemonstrative manner and reserved demeanor; evasiveness and irresponsibility; eagerness for being in the limelight and fear of being outdone, and evidence of dissatisfaction with the rôle of his sex. In the problems reported by teachers, shyness also heads the list, the comments ranging from "quite shy" to "painfully shy."

Reports as to the social adjustment for the 28 selected children enrolled in school show: 7 reported as leaders of their classmates; 15 accepted by their group, but described as followers, and 6 unpopular or isolated from the group.

Environmental Factors.—1. Ordinal Position in the Family: Hurlock and Burnstein [17] reported that: "Apparently the size of the family does not influence the creation." Only children were not, they found, more subject to the experience. The findings of this study indicate that although the phenomenon is, it is true, encountered in families of all sizes, the important consideration is the size of the family at the time the companion was created. Fifty-five per cent of the selected children were only children at the time of the creation of their companions. To check further, the selected and the control cases were compared from the standpoint of the percentage of only children in the two groups at the time of the fourth and fifth birthdays, respectively. The difference of the percentage is 25.8 to 6.0, and 15.6 to 5.2, suggesting again that the absence of siblings may be a factor. Chronological proximity to siblings appears, likewise, to be of some significance. The mean difference in age between the subjects and the next oldest siblings was five years and four months for the selected cases as compared with three years and one month for the controls; similar figures for the difference between the subjects and the next youngest siblings are three years and eight months, as compared with two years and seven months. Yet instances were found of wide chronological age-range between siblings without the occurrence of this phenomenon; likewise, there were cases of close proximity to siblings (less than two years) in which imaginary companions were created. Other factors obviously operate.

2. Opportunities for Companionship: Analysis of the data secured from mothers reveals that in 24 of the 40 cases opportunities for companionship were limited prior to enrolment in kindergarten, which is not possible in this community until the child is 5 years of age. In the remaining 16 cases opportunities for play with other children were not lacking. When companions were created despite the availability of real companions, there was reported a desire for more frequent or more satisfactory companionship. There was clear indication that many of these children were having difficulties in social adjustment, but whether these were greater than in the average child is not known.

In comparing the selected and control groups from the standpoint of opportunities for companionship particular attention was paid to the period between 2 and 3 years. While only 13 of the selected children had any consistent companionship before the age of 3 years, 32 in the control group had regular companions at this time. By the fourth birthday, 39 of the control group had daily companionship, while this was true of only 24 in the selected group. There was thus a conspicuous difference in opportunities for companionship in the two groups, both without and within the family circle. Yet not all children who voluntarily play alone or are isolated by the circumstances of their environment develop imaginary companions of the type under consideration.

3. Comparison of Siblings Who Did, with Those Who Did Not Have the Experience: Not only were the selected and control groups compared, but an attempt was made also to study children in the same family who did and who did not have imaginary companions.

Data are not available in regard to the comparative intelligence ratings of siblings who did have, and who did not have, imaginary companions, but a study of differences in the social situation indicates that the difference in age between the child and the next oldest sibling may be of some importance, the difference in the case of those who had imaginary companions being five years and four months, as compared with three years and three months for those who did not. The reports from mothers point to possible constitutional differences rather than to conspicuous differences in opportunities for companionship, those who had imaginary companions being regarded as more timid in their approach to other persons. It is clear that a more refined study of individual differences in intellectual, emotional and perceptual responses in childhood is indicated to determine why some children have this experience and others do not.

4. Parents' Attitude Toward Imaginary Companions: In only 1 of the 40 selected cases did the mother express alarm concerning the imaginary companion. The anxiety was based on the fact that the child was such an "awful liar." Two other parents recalled being somewhat concerned about how the phenomenon should be handled. One definitely discouraged it because it was used for purposes of evasion. In 36 cases it was accepted as a matter of course or even encouraged. The principal reasons advanced for encouraging this type of play were the pleasure afforded the child and the amusement it offered the parents. Few parents recognized that the phenomenon has any significance beyond that of substituting for real companions when these are lacking. From contact with approximately 800 mothers in this community during the period when the cases were selected, it was observed that the experience is less likely to be regarded as harmful by those who have witnessed it in the case of their own children than when it is a phenomenon that has never been encountered.

Data About Imaginary Companions.—1. The Approximate Age at which Imaginary Companions Appear: The fact that the subjects in this study were children rather than adults, 31 of the 40 children

selected being under 10 years of age, made possible fairly accurate data in regard to the age of the appearance of this phenomenon. In 37 cases the imaginary companion appeared before the fourth birthday, in 39 before the fifth, and in every case before the sixth birthday. The median age of appearance in this group was 2 years and 5 months. Although Hurlock and Burnstein [17] placed the most likely age for girls as from 5 to 7 years, reporting that with boys it appears somewhat later, Terman,[12] Norsworthy and Whitley,[9] Hall [4] and Kirkpatrick [6] reported observations that substantiate my observations.

2. Origins, or the Occasion of First Appearance: The finding of Hurlock and Burnstein [17] that the occasion for first appearance is often indefinite was substantiated by this investigation in both the children and the parents. Even in the 11 cases in which it was still active, exact data could not be secured. One reason why the memory of it may be obscured in the parents is that it is only after the companion has been mentioned repeatedly that he comes to have any significance for them. Yet some persons, as Hurlock and Burnstein discovered, recall the first occurrence.

One bright boy, aged 8, reported: "All I remember is that I was playing in the snow and all of a sudden this idea came to me and I ran into the gangway and whispered 'Ben Hur' (the name of the imaginary companion) into mother's ear." Unfortunately, data in regard to the immediately preceding experiences are not available.

Hurlock and Burnstein referred to lonesomeness, a feeling of being unjustly treated and the reading of stories as precipitating stimuli. Harvey [5] believed that there can be no supposition of conscious purpose in the greater number of cases.

Although suddenness or unexpectedness may characterize the first appearance of an imaginary companion, later appearances seem to occur at the child's will, to judge from the following statements taken from the interviews with the selected children:

I wrote them a letter last night and told them to come.

I don't want my boy to come; he minds me. (This was a manifestation of resistance to being questioned about the imaginary companion.)

I used to talk to them when nobody was in my room.

3. Names of Imaginary Companions: The names given imaginary companions may be commonplace (these predominate) yet they are often highly original, as the following collection reveals:

Gengerā'les
Kātsch
Tăgar'
Bĕr'ry and Auntie
Bōr'ăc Sĕlēē'to
Mā'vee
Jăck'son Sēē'gar
Sister Mĭg Măn
Darn
Doy'da (oi)
Hĭm'lay and Gâr'gla
Hill and Hell
Ave and Dēēve
Chopsticks
Dō'dō and Curly Stockings

The following are additional names given to imaginary companions by children not included in the study.

Mĭńjŭn and Făl'ley
Mrs. Băl'bun
Ĕk'rons
Bing and Bung
Bŏm'bean
Tŭb'ba
Blŭb'bin
Mrs. Gōpĭk'ēē
Mississippi
Hĭnje and Mĭnje
Dā'tō and Jāz'o
Pīk'ee
Bäh, Fūr and Sĭx'en

Original names tend to be simple vowel and consonant combinations in words of two or three syllables, combinations easily articulated and characterized by alliteration, rhythm and rhyme.

An analysis of the sounds shows certain recurring patterns. The bilateral consonants p, b and m, and the guttural sounds k, g and ng, are those most frequently encountered. They are the earliest sounds learned in the course of normal speech development. That the bilateral consonants frequently appear is doubtless because the movements of articulation are observable. The guttural sounds the child stumbles on accidentally in gurgling. The only vowel which reappeared consistently, e, was most prominent at the end of the name.

Evidence points to the following as possible sources for the names of imaginary companions: (1) real characters, particularly those loved or admired; (2) persons or words which are surrounded with mystery or whose meaning or relationship is not fully comprehended by the child (when this is the case, the sounds may be inaccurately reproduced, as when Adam and Eve became Ave and Deve); (3) children's stories, and (4) euphonious sound effects.

4. Characteristics of Imaginary Companions: There was no evidence of a uniform course of development for the phenomenon. The first consistent character was, in four instances, an animal. The majority of the earliest creations were, however, persons. Of those created at a later age, some were animals. The first imaginary companion or companions, for they often appear in pairs, are apt to give way to new creations, the original character being dropped completely or played with simultaneously. This was reported to have occurred in 13 of the 40 cases.

Although in 8 cases the companions were created in pairs, in only 3 were imaginary families reported. There was evidence from interviews with the children that elaboration along family lines occurs more frequently than parents are aware. One bright girl, with an intelligence quotient of 160, developed the imaginary family between the ages of 2 and 3 years. In the other cases the complex family structure did not appear until the fourth and fifth year.

The exact nature of the personality of the imaginary companions and the child's relationship to them appear to be socially determined,

and consequently vary with each case and from time to time. For this reason it is essential that the phenomenon be interpreted always against the background of the specific social situation, particularly the child's social relationships.

There is evidence that when imaginary companions are conceived of as having ability or status superior to the child creating them they are merely referred to. Those who are addressed directly usually occupy a subordinate rôle.

In 33 of the 40 selected cases the most vivid imaginary companions were of the child's own sex; in 6 they were of both sexes and in only 1 were the companions of the opposite sex solely. This was the case of a girl, aged 3, reported to have a stern father.

In 18 of the 40 cases the exact relationship of the imaginary companion was definitely indicated by the child itself: seven were labeled "sister" or "brother"; one was called "my boy"; one was called "Aunt Grace"; one was called "Bill, my husband," and eight were designated "friends."

Of the 22 cases in which it was necessary to rely on the mother's impression, 20 of the companions were viewed as friends, only 2 as siblings. The rôle of friend, therefore, appears to be the most common.

Though more likely to be of the child's own age, the imaginary companion may be of an indefinite age, combining the characteristics of adult and child. "Katsch," for example, was at one time "a big lady like you, mamma," but at another, a child ordered in from play. The child was consistently in a superior position in relation to "Katsch," however.

One mother, whose daughter at the age of 3 years had an imaginary sister, regarded the phenomenon as distinctly pathologic, because her daughter became ill with dementia praecox at 17 years of age. From the account given by the mother the nature of the play in this case was not essentially different from that of other children who had imaginary companions. The mother commented, however, on the fact that the imaginary companion was discussed more freely outside the home than within the family circle.

5. Play Activities with Imaginary Companions: Thirty-three of the 40 selected children played with the imaginary companions in such a manner as to indicate that the companions were conceived of as occupying space. They were spoken to directly, were chased in games or were brought to the table where a place was set for them. The exact form which the play takes appears to be directly related to the child's own experiences, and therefore varies from case to case. Yet certain common features of this play can be observed. Activities shared with imaginary companions are usually those which are highly charged emotionally by virtue of being novel and pleasurable, or

humiliating and consequently painful. The play reflects parental attitudes, particularly disciplinary attitudes and the child's reaction to them.

Imaginary companions live elsewhere than in the child's home, even when they are conceived of as siblings. The exact location, if definite, is usually significant. For example, Ann's "boy" lived in Homewood, the town in which her nurse-maid lived and to which the nurse-maid returned each evening. "Boy" was always doing everything that Ann was doing, but in Homewood.

Contrary to the findings reported by Harvey,[5] the location of the abode of the imaginary companions causes the children concern. Often during walks they announce an intention of visiting them and designate one or another house as the home of the fantasy character.

6. Sharing of Imaginary Companions: In all the 40 cases included in this study the imaginary companions were talked about freely in the family circle or within the hearing of siblings or parents, yet belonged essentially to the child creating them. In only 3 cases were they actively shared with siblings and in only 5 cases were they discussed or shared with real playmates. This sharing had unhappy consequences for one child, who was disturbed because her playmates did not understand about "Katsch." One child, a boy, aged 5, took his playmate with him to kindergarten and announced his presence to the teacher.

7. Disappearance of Imaginary Companions: Anything approximating accurate information in regard to the time at which imaginary companions disappear is difficult to obtain, owing to the gradual character of the process. There is evidence, moreover, that they are played with secretly after such time as the child becomes aware of social disapproval. They may, then, take on a more definite make-believe character.

According to reports from mothers, the imaginary companions continued to figure actively in the play life in only 11 of the 40 selected children at the time of the investigation. These children ranged in age from 3 years to 7 years and 9 months. Conversations with the children revealed, however, that they were still played with in 16 cases. The oldest of these was a boy, aged 9 years and 11 months, who played games with the companion during the recess periods at school.

Reports from mothers show that of the 28 children whose parents said the imaginary companions had disappeared, 22 had ceased to talk about them openly by the sixth birthday and 18 before the fifth. Disappearance was credited, in the 22 cases in which it was an experience of the past, to increased opportunities for companionship, usually enrolment in kindergarten at 5 years of age. Hall,[4] Tanner [11] and Harvey [5] likewise related the disappearance to more satisfactory opportunities for companionship.

One case included in this study serves to illustrate that there are factors other than increased opportunities for companionship or ridicule which bear on the disappearance of this phenomenon.

A boy, 3½ years of age, four years younger than the other child in the family, an aggressive girl, created his imaginary brother at 2 years of age. The mother, likewise an aggressive person, compared the boy unfavorably with the father's family. She was irritated by his timidity and the fact that he did not respond to the training methods employed with his sister. Incidentally, this boy had excellent opportunities for companionship and played outdoors daily. The mother noticed that the imaginary companion seemed to belong exclusively to the child; consequently she paid little attention to it until she learned of the present investigation. Suddenly she became interested. The result was that the "brother" was never mentioned again.

8. Reality of the Imaginary Companions: There was no check on the reality or unreality of the phenomenon other than the children's statements. Despite the lack of uniformity in the interviews, which were necessarily individual with so personal a phenomenon, one notes a certain similarity in responses at given chronological ages and a gradual transition to the point at which the make-believe character of the phenomenon is clearly recognized. One girl had definitely established the make-believe character of the play by 4 years and 9 months of age. In general, the period from 5 to 6 years appeared to be the transition period. Such remarks as "They're crazy things" from a child aged 3, and "In my heart I can see him," from one aged 5, point to the fact that children themselves draw some distinction between imaginary and real companions, possibly from the beginning.

Leaving aside the question of origins, there is clear indication that the experience is accompanied by visual imagery. In 17 cases statements descriptive of the appearance of the companion were secured.

9. Child's Attitude toward Questioning about Imaginary Companions: Something of the attitude toward the phenomenon can perhaps be deduced from the child's reaction to direct inquiry about it on the part of a stranger. In every instance, except when the child was more than 11 years old (then a frank explanation of the study was made), the approach was made in this way:

"Do you sometimes play with————?" If resistance was encountered, the investigator went on to say, "Mother told me you played with————." If necessary, she explained that she herself had had imaginary companions. The following types of response were noted in the children who were interviewed:

Five children greeted the initial question with smiles.

Sixteen children took the question seriously and responded in the same manner.

Three children denied recollection of it at first, steering the conversation to real companions, but later cooperating by giving some details (age range: 4 years and 9 months to 6 years).

Two children denied any connection with it, charging siblings with the possession of the companion (1 of these cases was still active).

One child was decidedly apprehensive. An initial affirmative response from this 6½ year old girl was followed by "I don't know what you mean." Further questioning was increasingly resisted.

SUMMARY

Vivid and sustained imaginary companions were reported for 13 per cent of the children in control families. They were encountered in this study three times as often among girls as boys. In four families they were reported for 2 children in the same family, widely separated in chronological age.

The phenomenon is not limited to children of superior intelligence, though apparently more prevalent among them.

Personality difficulties in some form were reported for 35 of the 40 selected children, timidity heading the list. Seven of the selected children were, however, described as leaders.

The phenomenon is encountered in families of all sizes, but at the actual time of the creation of the companions, 55 per cent of those selected were only children. Chronological proximity to siblings appears to be of some significance, the children who have imaginary companions tending to be farther removed from siblings.

There was a conspicuous difference in the opportunities for companionship in the selected and control groups, particularly between the ages of 2 and 3 years.

In 36 of the 40 selected cases the phenomenon was accepted as a matter of course or even encouraged by the parents. Few parents recognized that it had any significance beyond that of substitution for real companions. There is evidence that the phenomenon is more likely to be regarded as a possible harmful manifestation by parents who have not encountered it in their own families than by those whose children have experienced it.

The median age for the appearance of the first imaginary companion was in this group 2 years and 5 months. Although there are marked individual differences, the period from 5 to 6 years may be regarded as one of transition to the point at which the make-believe character of the play is definitely acknowledged by the child.

The names given to imaginary companions may be commonplace (these predominate), yet are often highly original. Possible sources of names are: (1) real characters, particularly those loved or admired; (2) persons or words surrounded with mystery; (3) children's stories, and (4) euphonious sound effects.

In 33 of the 40 selected cases the most vivid imaginary companions were of the child's own sex. Though more likely to be of the same age as the child, they may be of an indefinite age, combining the characteristics of adult and child. While they were frequently labeled relatives, the rôle of friend appears to be more common.

The exact nature of the personality of the imaginary companions and the child's relationship to them appear to be socially determined, and consequently vary from case to case. There is evidence to point to a tendency to address directly the companions who occupy a subordinate rôle, those superior in status being merely referred to.

Activities shared with imaginary companions are usually those which are highly charged emotionally by virtue of being novel and pleasurable, or humiliating and consequently painful. The play tends to reflect parental attitudes, particularly disciplinary attitudes and the child's reaction to them.

There is clear indication that the experience is accompanied by visual imagery. In 33 of the 40 selected cases the imaginary companions were played with in such a manner as to indicate that they were conceived of as occupying space. In 17 instances statements descriptive of the appearance of the companion were secured.

APPENDIX

Suggested questions for the further study of the phenomenon of imaginary companions are:

1. What are the possibilities of investigating this phenomenon by exact laboratory methods?

2. What is the relationship of the phenomenon to eidetic imagery?

3. What are the factors which contribute to individual differences in the quantity and quality of imaginative activity? To what extent can imagination be considered as a separate factor, or is it the result of various intellectual and emotional factors (what is the relationship to unsatisfied emotional needs)?

3. Does the phenomenon of the imaginary companion correlate with a general quality of imaginativeness?

5. Of what significance are parental and sibling relationships for the phenomenon of imaginary companions?

6. To what extent is the tendency to remove oneself from one's concrete situation by the assumption of another rôle related to emotional stability or instability? What are individual differences in this connection, and the differences between children and adults?

7. To what extent is this tendency to disassociation related to constitutional factors or a learned reaction?

8. What common defects in adjustment do children who have imaginary companions show besides timidity?

9. Would investigation and long-time observation reveal any correlation between the phenomenon of imaginary companions and the development of homosexuality, schizophrenia or hysteria?

10. To what extent is the phenomenon of the imaginary companion related to artistic ability and interest?

11. What is the function of imaginary play?

PSYCHOSES ASSOCIATED WITH SOMATIC DISEASES THAT DISTORT THE BODY STRUCTURE

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Somatic diseases that distort the body structure cause an insult to the physical personality which the subject finds difficult to accept. The pathologic process results in a discrepancy between the body structure and the body image constitutionally and socially acceptable to the patients. This study attempts to show how the psychosis which may arise in such persons is due largely to this discrepancy and represents, in part, a specific psychopathology related to disturbances in the body image, postural model or body schema.

The concept of the postural model was first introduced by Henry Head.[1] To him, however, it meant merely the integrated concept of the body posture in gait and station, which is constantly changing as the gait and station change, through the peripheral sensations that arise from the moving or static body. "Man perpetually builds up a model of himself which constantly changes. Every new posture or movement is recorded in this plastic schema, and the activity of the cortex brings every fresh group of sensations evoked by altered posture into relation with it." Schilder [2] has developed a much more comprehensive concept from his earlier work on the *Körperschema*,[3] built on "the mechanisms of the central nervous system which are of importance for the building up on the special image everybody has about himself." This study is based on physiology, neuropathology and psychology, including Schilder's psychoanalytic experiences. He has incorporated this material into a psychologic doctrine with far-going implications. The essentials of the doctrine are that there is a plastic concept of the body image which is built from all sensory and psychic experiences and is in constant integration in the central nervous system into a concept of *Gestalt* which "sees life and personality as a whole" and which may become variously modified and distorted by lesions in the central nervous system or by pathologic conditions in the psychic levels of the personality.

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1. Head, Henry: Studies in Neurology, New York, Oxford University Press, 1920, vol. 2, p. 669.

2. Schilder, Paul: Image and Appearance of the Human Body; Studies in the Constructive Energies of the Psyche, London, George Routledge & Sons, Ltd., and Kegan Paul, Trench, Trubner & Co., Ltd., 1934.

3. Schilder, Paul: Das Körperschema, Berlin, Julius Springer, 1932.

The body image is something more than a postural model arising from the changing body movements and positions; it is also something more than an integrated pattern of all the immediate organic and psychic experiences. In some way it preceded and determined the individual body structure. It is the pattern of the evolutionary species predetermined in the biologic matrix. The pattern of the individual body image is determined biologically, constitutionally and by direct heredity. This is also a part of the concept of the body image experienced by every one. It is perhaps the most fixed and primordial part of the concept. The pattern is laid down in the anlage of the embryo through the pattern of the growth tendencies. Coghill [4] has shown this in his developmental studies of Amblystoma, published in his work on "Anatomy and the Problem of Behavior." He has shown that "all behavior develops from the beginning through the progressive expansion of a perfectly integrated total pattern." [5] He has shown also that the integrated pattern of the total behavior of the subject exists already in the preneural mechanism as well as in the developing nervous system. He then claimed that "the maintenance of the integrity of the individual as a whole is the elementary function of the nervous system." [6] "Man is a mechanism which is creating and operating himself" by growth patterns which are integrated from the beginning. Of course, in keeping with Schilder's teachings, the concept is constantly modified by new experiences as well as by new predetermined stages in the development. Still the fundamental basis for the body concept is in the inherent integrated pattern and its growth potentialities. This is dependent on biologic and constitutional laws. It determines the static background for the body image. It also determines the body structure and the physical personality. There should be no discrepancy between the body image and the body structure, but accidents or diseases may modify the body structure in ways not planned and anticipated by the body image. The body image may be slow to respond to these discrepancies.

In addition to this primordial or constitutional concept of the body image there is a concept of the body image that is socially determined. One sees and otherwise experiences the body image of other people. There are many variations in body types, but one becomes acquainted with these and in one way or another identifies himself with them so that they become a part of one's own composite body image. A rare and obscure disease which modifies one's own body structure in a way that has not been seen or experienced in any one else is difficult to

4. Coghill, G. E.: Anatomy and the Problem of Behaviour, New York, The Macmillan Company, 1929.

5. Coghill,[4] p. 38.

6. Coghill,[4] p. 90.

conceive and tends to create a pathologic discrepancy between the constitutionally and socially accepted concept and the actual body structure.

One's final view of the body image or the *Gestalt* of the physical personality is: (1) predetermined by an integrated pattern biologically established by laws of growth and constitutionally fixed; (2) socially recognized and accepted by contact and identification with other physical personalities; (3) constantly modified and reconstructed by the actively integrating nervous system that utilizes all the new experiences of the sensations and the psyche (Schilder), and (4) immediately modified by the body movements and positions (Head).

Diseases in any part of the sensory mechanism that records movement of posture may disturb the postural model of Head. Diseases in the nervous system or in any part of the sensorium or psyche may disturb the *Körperschema* and give rise to the great number of disturbances described by Schilder.[2] Slowly developing or chronic diseases that distort the body structure, either immediately or by interfering with its growth, tend to create discrepancies between the body image and the body structure. The fundamental features in the psychopathology of such a condition depend on: (1) the actual conceptional difficulties in accepting or recognizing the discrepancies, or what Schilder refers to as imperception or anosognosia, which lead to specific psychopathologic symptoms, (2) discrepancies of behavior arising from the thwarting of the anticipated adult social adaptation which would have been appropriate for the expected and accepted body image but which is not possible for the given body structure, which lead to emotional symptoms, (3) failure to comprehend the nature of the disease, with feelings of mysterious persecutions, which leads to paranoid symptoms and (4) coincident symptoms dependent on toxic features of some of the diseases, or pressure of the distorted bony structure on the nervous system, which may lead to confusional symptoms or exaggeration of any of the preceding symptoms.

The groups of diseases I shall report include diseases of the bony framework that distort the structure of the skeleton, such as osteitis deformans and osteogenesis imperfecta, diseases of growth, resulting in defective growth or dwarfism and diseases of large sectors of the nervous system that regulates motor and sensory function of the body, such as Little's disease, poliomyelitis anterior and subacute combined sclerosis.

OSTEITIS DEFORMANS (PAGET'S DISEASE)

Osteitis deformans or Paget's disease is not uncommon but is comparatively rare. It is defined by Knaggs[7] as a "very chronic disease

7. Knaggs, R. L.: Diseases of the Bone, New York, William Wood & Company, 1926.

of an inflammatory nature, occurring in middle-aged or elderly people, in which the normal osseous structure of the skeleton is removed and fresh bone formed on a different plan and in a larger mould takes place." Clinically, it is characterized by gradual thickening and softening of the bones, especially the long bones and the skull so that there are bowing deformity of the legs and a convexobasic deformity of the skull, with consequent pressure on various parts of the brain or emerging cranial nerves. It may be associated with neurologic signs, headaches, epileptiform attacks and visual, auditory and sensory disturbances. Sometimes there is loss of teeth. Studies of the brain are rare. Grünthal [8] reported a case with cerebellar signs, due to compression in the posterior fossa, which showed atrophic loss in Purkinje cells and in the dentate nucleus with glial replacement. Although Paget [9] himself said: "even when the skull is hugely thickened and all its bones exceedingly altered in structure, the mind remains unaffected," more than a score of cases of psychoses in Paget's disease have been reported in the literature [10] and in the past two years eight patients with Paget's disease have been admitted to the psychiatric division of the Bellevue Hospital with some type of mental disturbance. The literature has shown little effort to interpret the mechanisms of the mental disturbance as a response to the severe somatic insult to the physical personality.

CASE 1.—L. DeG., a mulatto woman, aged 61, single, a dressmaker, who was brought to the hospital from the Municipal Lodging House complaining of pains in her legs, presented the typical features of osteitis deformans. She was short in stature, with saber-shaped, bowed legs and a large misshapen head, which was enlarged especially through the frontotemporal diameter. The eyes were deep set; the temporal vessels were prominent, tortuous and pulsating. She was deaf and had the high-pitched, poorly modulated voice of a deaf person. Gait was awkward and straddling. Examination of the pupils, eyes, vision, and nervous system gave negative results. The internal organs were normal, except that the blood pressure was 200 systolic and 110 diastolic and there was peripheral arteriosclerosis. The teeth were practically all missing, except for a few decayed stumps. The Wassermann reaction of the blood was four plus; serologic tests of the spinal fluid gave negative results. Roentgen examination showed: "Paget's disease involving the bones of the skull and the long bones."

Mental State.—The only history available was that given by the patient. She said on admission: "I had painful legs and wanted to have them examined. I

8. Grünthal, E.: Ueber den Hirnbefund bei Pagetscher Krankheit des Schädels, Ztschr. f. d. ges. Neurol. u. Psychiat. **136**:656, 1931.

9. Paget, James: Lancet **2**:715 (Nov. 18) 1876.

10. (*a*) Kaufman, N. R.: Psychosis in Paget's Disease, Arch. Neurol. & Psychiat. **21**:828 (April) 1929. (*b*) Schiller, M. A.: A Case of Paget's Disease with Psychosis, ibid. **27**:226 (Jan.) 1932; abstr., ibid. **29**:203 (Jan.) 1933. (*c*) Smith, L. H.: Psychoses Associated with Osteitis Deformans, J. Nerv. & Ment. Dis. **68**:578, 1928. (*d*) Stauder, K. H.: Psychic Disturbances in Osteitis Deformans, Arch. f. Psychiat. **98**:546 and 566, 1933.

don't hear very well. I am a good dressmaker and work for the school of dressmaking. I can't stay in a hospital because I have to go to school. I have been going all these years, and I don't want to quit. I just want to have my legs looked at and go right home. Those bumps on my head are due to classes experimenting on me. They took all my teeth, too. My head used to be much worse, but it flattened out. But it was my teeth I minded the most. I had beautiful big white teeth, and when I woke up in the morning I would spit them out, two and three at a time. I blamed the woman I was living with. She didn't let the classes experiment on her children. They only experimented on me. I could hear them talking, too, but I could not tell what they said except once. I heard a woman's voice say, 'Her profile isn't perfect.' But that was their fault. When they pulled out my back teeth my neck line fell in. I couldn't even see them. If I could only have seen them I could have made them stop."

Later, she explained the ideas more in detail. "Whatever happened to my head, I thought the classes did it. I never could forget it because the pain in my head was something terrible. But I couldn't complain because I was not in my own house. I thought that they were all her friends that fooled with my body. Her crowd pulled my teeth out. I heard them in the night talking about my teeth. I had fallen asleep, and in the morning I had found my teeth missing. I didn't think that was so funny. Just think of it. I bet all her children have all their teeth. I didn't have any one to help me. They were her friends. I heard one say one night, 'Well, I want to learn all I can.' They pulled my face all to pieces, just the same, to learn on my face. Any one can come along and knock you in the face. I was going downtown in the train and they shoved me and said, 'We pulled out those teeth.' I had to sit there and take it. I got on the car one morning, and three or four were behind me. I didn't see them exactly, but I heard one girl say, 'I can't make you look like her. I am doing all I can, but I can't make you look like her,' and the other girl said, 'I don't think it is right either, to take her to practice on.' I wasn't bowlegged as a girl. That all happened since. This class was fooling with my body. I could tell it from the looks of my clothes that they were pulling my body to pieces. In here (hips) I am too broad. I was not shaped like that, I mean in the broad, big, ugly part. I don't know what they did to me but they did something to me. They keep me from looking nice in my clothes. I made a dress from my dressmaker's form and couldn't tell what in the world was the trouble with it when I went to put it on. I took a tape measure and measured myself and found quickly enough what the trouble was. I was so big it didn't look neat and nice at all. I didn't feel it, but I looked it. But when they fooled with my head, what terrible headaches I had! Oh Lord, what a pain! To get free from them I would run out in the street. Then I would be too long from my work."

The patient was retiring, dull and preoccupied. She was also somewhat confused and showed some intellectual defect. There was no queer behavior, catatonic features or dissociation in intellectual or emotional response. She was transferred to the Central Islip State Hospital on Nov. 11, 1932.

A report from that hospital shows that the physical status remained the same. The examination of the spinal fluid gave negative results although the Wassermann reaction of the blood was four plus. The mental status showed mental confusion, and the patient complained in the same way of classes of boys experimenting on her, stating that by means of electricity they put bumps on her head and made her teeth fall out. The deafness proved a handicap in her adjustment, as did the intellectual defects, which may have been partly dependent on arterio-

sclerotic changes in the brain, and she became increasingly withdrawn, suspicious and uncommunicative. She is still at the Central Islip State Hospital in a confused, negativistic state.

Comment.—This case presents a well organized delusional system based on somatic sensations and experiences arising from an obscure, slowly developing chronic disease which distorted the body of the patient. There were ideas of reference and some auditory hallucinations appropriate to the delusional system. The psychosis was further encouraged by deafness. The syndrome is typical enough of paranoid schizophrenia to justify this diagnosis, although it shows none of the organic features, such as catatonic features, dissociation of thought processes or inappropriate behavior. The mechanism of the psychosis can be understood from a knowledge of the patient's experiences.

This case is of particular interest as showing the disturbances that arise in the emotional life from mysterious changes to the body image or postural model, because the patient, as a dressmaker, compared her gradually changing body to the dressmaker's form which in some way represented her original postural model. Such a dressmaker's form is made to order from individual measurements and represents the body of the subject; henceforth, dresses are made to fit the form with the expectation that when finished they will fit the subject. The dresses for the patient, made from her form, did not fit; this caused her great consternation because she had not recognized the change in her body structure. By making measurements she discovered the changes that had distorted her body structure while her body image had remained in accord with her old concepts. It was as though she retained the fantom of her former postural model, of her body as she knew and loved it, and was unwilling to give it up. This, of course, was of special importance in this woman who had put so much narcisistic libido into her body, its form, the profile of her face and neck and her much cherished teeth.

CASE 2.—N. B., a single, white woman, aged 55, was brought to the psychiatric division of Bellevue Hospital on April 2, 1933, by a sister and a physician, who stated that for two weeks she had been excited and talkative, running about on the street and responding to imaginary voices. The history showed that she had studied in college for two years and had become a clerk; for ten years she had worked in the building department in the office of president of a borough. About eleven years before she had been struck on the head by a stone from a city building. She was not unconscious, but it was then first noticed that there was a bump on her head; from that time on the head had grown in size. Neither at the time of the accident nor at any subsequent time would she consult a physician, however, as she was urged to do. She complained that she was discriminated against in the office and was not promoted as rapidly as others. In February 1931, she believed that the name of a man who had died several months before was still being carried on the payroll, and she notified the civil service commissioner. As a result she was asked to identify a man as the supposed dead man; she said that when she denied the identity she was grabbed by the right arm by her superintendent and from that time on the arm was paralyzed. From that time on she complained of being constantly annoyed. For some weeks before admission she had become progressively more excited and said that men from the office were following her on the street and that she and her sister were going to be killed. She stayed at home and kept the window shades pulled down; finally she became panicky and claimed that she was to be electrocuted. She told the physician who was called that there were machines in the wall which talked to her and told her she was the "court stenographer."

Examination.—On admission the patient was excited and said, "They want to call me 'Court Stenographer.' People spit in my face, and the superintendent knocked me down. People insult me and blow smoke in my hair. What is going on?"

She was resistive to physical examination, and at no time allowed herself to be adequately examined. She would become very resistive at any effort to touch her. However, she presented features typical of osteitis deformans. She was short in stature, with anterior bowing of the arms and legs. There was a bony overgrowth on the left tibia. The patient said that she had sustained a fracture there. The left arm was weak and tremulous. Careful examination of this arm was not permitted, though there was evident paresis. The skull was enlarged and nodular. The teeth were misalined. Vision was intact, but hearing was somewhat impaired. According to the sister this had been evident only for the preceding few months.

When an effort was made to examine the patient she said: "You don't examine me. You don't do any examining. I go under insanity. You don't examine me. I don't belong in this ward, anyhow. I belong in the insane ward. You get no information from me. I didn't come here for nervousness. I came for—I came with my own doctor to have my head examined. See if I am insane. They said I hear machines, and if I hear machines I am insane. Mysterious machines. They seem to be floating in the air. I imagine I hear voices. I would have to have a brain as big as this room if I told you all the things they said to me. O don't remember—that is my fault. Yes, people have been trailing me. I don't know why—maybe I was only imagining it. I never counted them. I would have some time counting up all the people that trailed me. I don't know how long they are after me. I didn't keep any record. No, I don't see visions, but I hear voices—I imagined I hear the machines. They tell me to do things. They seem to carry on a conversation and tell me things. Yes, they wanted to change my title. They called me a 'court stenographer,' these mysterious voices. Yes, the people on the street carry on a conversation, too. I've been sick for some time. I work occasionally. My head has been big for some time. This arm is hurt about two years."

She became increasingly resistive, disturbed and noisy, requiring medication for sedation, restraint and tube-feeding. At intervals she said, "I am here because the gas was turned on by some one who wanted to kill me. Now it is up to the detectives to find him." At another time she said, "I am in conference with the Governor of New York. Don't touch me. He says you are to leave me alone. No, this isn't wireless, but it is something like it. Don't interrupt me; it is very rude. I will tell you all about it when I have finished, if you will let me alone now."

Course.—The patient was transferred to the Central Islip State Hospital on April 7, 1933, and continued to be disturbed, resistive and struggling; she threw herself out of bed and sustained a skull fracture. She was rambling in speech. She claimed that for two or three weeks she had been in communication with Governor Lehman, Jimmy Walker and others, and that they talked to her and she answered them. She said that a clique had been following her for about seven or eight months, owing to the trouble she had had at her place of work for the past two years. Pulmonary congestion developed and she died on April 10 as a result of the skull fracture and cerebral hemorrhage.

Comment.—This case presents a slightly complicated mechanism which, however, can be readily understood by the combination of the patient's experiences.

For seven years she had had a mysterious disease of the body which she would not openly recognize, and for which she would not consult a physician. It seemed to her to be associated with a stone falling from a city building. Subsequently, as a result of her own feelings of being persecuted, she noted an error on the payroll and became involved in a situation that led to considerable suspicion and gave her reason to think that she was being persecuted. In the course of this she received a relatively minor injury that led to an injury to one arm. Later, as her hearing became impaired and noises in the head developed, she became acutely excited, hallucinated and deluded. The content of the psychosis was concerned with personal problems and the strange condition of the body. "If I hear machines in my head I must be insane. They seem to be floating in the air (vestibular phenomena probably arising from pressure on the eighth nerve). I would have to have a brain as big as this room if I told you all the things they said." Her death is psychologically a suicide and, again, comes from a relatively minor injury to the diseased skull, as well as from the marked resistiveness and unwillingness to have any one handle her distorted body.

The same unwillingness to recognize the disease and the disabilities caused by it is seen in the following case. Here, it is apparently an actual imperception or anosegnosia for the defect.

CASE 3.—M. M., a widow, aged 61, was transferred to the psychiatric division of Bellevue Hospital on Nov. 10, 1932, from the City Hospital where she had been since 1926 because of a crippled state due to Paget's disease. For many years she had been unable to walk without crutches. Information as to the past history could not be obtained, however, except that the records of the hospital showed that the patient had had pains in and bowing of both legs and had been becoming shorter fifteen years before admission. She was transferred to the Bellevue Hospital because she was becoming "extremely obscene"; she called the nurses "street walkers," but usually used less euphonic language. She stopped the doctors to tell them about spanking her cat for staying out all hours of the night, although she had not had a cat in the five years she had been in the hospital. "She scratches and bites whenever the nurses try to attend her."

Examination.—Detailed physical examination was not permitted by the patient. She fought, scratched and bit in the most vicious manner whenever an attempt was made to touch her body. As she was helpless to care for her personal needs, daily nursing care proved to be a great problem. Obviously she was suffering from osteitis deformans, with such marked bowing deformity of the arms and legs that she was fixed in a sitting posture with the arms and legs before her. The skull was enlarged, nodular and flattened on the vertex. She was totally blind, although she did not admit this and was very cunning in evading tests to prove it. The pupils did not react to light. An unsatisfactory view of the optic disks showed optic atrophy. Hearing, however, was intact. The heart was enlarged, with mitral systolic and diastolic murmurs and an aortic systolic murmur. The Wassermann reaction of the blood and spinal fluid was negative. The urine contained albumin (two plus). The blood chemistry was normal. Roentgen examination showed "configuration and consistency of the bones of the skull characteristic of Paget's disease."

Mentally, there was some defect of memory which the patient did not or would not recognize. She was not hallucinated or deluded. At all times she was irascible, irritable and obscene, and defended herself against an unfriendly and inquisitive world. When asked how long she had been crippled, she said: "None of your business. Go to hell and get out of here. You won't find out anything

from me. Leave me alone, God damn you." (Do you know where you are?) "I know where I am dammed well; now get out of here." (Won't you shake hands with me?) "No, I won't give my hands to anybody." (Can you see well?) "I have lovely eyesight, thank you." (What is this?—showing a key.) "It is a hundred dollar bill—now aren't you smart? You know darn well what it is." (How many people are there in this room?) "There are one hundred and forty-five, my way of counting." (But you didn't count them.) "I don't have to." (What brought you here?) "I don't know. I don't bother with any of the nurses. I just go ahead. Don't be talking to me. You give me a headache. Everybody talks. Everybody says one hundred and forty-nine things. Now don't bother me. Will you please leave me alone? I don't know the name of this place and I don't want to. They are all alike, these hospitals. They aren't much good. They all treat you alike. Every one for themselves. I declined letting them do anything for me. I would hit them." (Tell us about your cat?) "I had a cat named Chopsy. He ran away on me. He was black and white. Sometimes it is true I get cross, but I never use bad words. I have been a very good girl. Hospital life is hard life. I wouldn't like to tell you how long I have been in hospitals. You make my head so bad. You talk too much. I have no use for the nurses. I wish you would quit asking me questions. You will have your head fractured. There is nothing the matter with my head. It has been the same way for years. I am a cripple, but my legs have always been this way. I couldn't tell you how long."

The nurses reported: "The patient sits up in bed with her legs crossed. She is incontinent and apparently blind. When touched she screams and curses, 'You s.—o.—b.' She is uncooperative and abusive. She complains of muscular pains and headaches. She is depressed and pathetic-appearing."

Course.—The patient was transferred to the Manhattan State Hospital on Nov. 11, 1932, and died on November 30 of pneumonia without any change in condition.

The following case also presents some problems that arise when the patient attempts to ignore the disease, and is unable to accept it. This case also presents other problems related to the psychopathology of disturbances in the postural model.

Case 4.—C. B., a man, aged 52, who was transferred from the surgical to the psychiatric service of Bellevue Hospital on Oct. 27, 1933, had been treated for two weeks for a broken leg, but had become progressively more restless and uncooperative, repeatedly removing the bandages and traction apparatus from his leg and beating his face and head in the night with his fists. It was obvious that he had osteitis deformans, as the head was enlarged and nodular in the typical way. However, he denied any knowledge of this to the doctor. "No, there is nothing the matter with my bones or my head. I haven't bought bigger hats lately. I haven't bought any new hats at all lately. I don't understand why my bones break so easy. I was over at Polyclinic Hospital last year with a broken arm. I got depressed there, too. I can't stand not being on my feet and active. I don't remember beating my head. Maybe I did beat it unconsciously in my sleep. I'm so nervous, I've had so much trouble. Oh God, I wish I was dead; I break all my bones."

Roentgen examination showed Paget's disease of the skull, pelvis, femur, humerus and forearms. There was also a fracture of the midshaft of the right humerus, with deformity and a transverse fracture of the upper third of the right femoral shaft with displacement.

Course.—The patient continued restless, complaining and uncooperative; he moaned and cried most of the time, especially at night. He would say, "I wish I was dead. I have so many reverses. Nothing seems to go right. Do cases like mine ever get well?" Although he denied knowledge of his disease to the doctors or nurses, he was overheard telling another patient on the ward that he had Paget's disease, which is incurable. He would often pray that he might die, saying that he was the most uncomfortable man in the whole world. His tendency to deny his troubles was at one time limited to the offending leg. He said, "This is not my leg. It belongs to the other fellow." He then beat himself in the face, called for help and attempted to choke himself, saying that he would not get any better and wanted to die. He was transferred to the Manhattan State Hospital on December 8, still restless, depressed and complaining.

Comment.—In this case of Paget's disease the patient tried to deny the existence of the mysterious, incurable disease that causes fractures so readily and thwarts all personal efforts to get ahead in the world. He berated himself, pounded his diseased head, and pulled the tractions from his leg, but said that he was "unconscious" when he did these things. At one time he denied the existence of the broken, diseased leg, saying, "It belongs to the other fellow." This failure to perceive a defect and the tendency to deny that an affected part of the body belongs to one are discussed by Schilder [2] in his book, "Image and Appearance of the Human Body," as a psychic process in which the patient consciously or unconsciously does not wish to acknowledge the defect, and therefore represses it. This process seems to be true in this case.

In case 3, the failure of the patient to recognize her blindness may lie at the organic level of depression. In this case, the optic apparatus was involved specifically, and in a more general way the brain was undoubtedly distorted by the deformity of the skull, as discussed by Grünthal.[8] The patient showed evidence of defects of memory and confusion which may also be associated with her age and with the arteriosclerotic changes. Anton, quoted by Schilder, related this nonperception of the organic level to localized cerebral lesions. However, even in case 3, some of the nonperception and repression of recognition of the patient's condition and her active defense against inquisitive queries seemed to be at a psychic level.

That the psychopathology of Paget's disease is at least partly connected with the feeling of mysteriousness and hopelessness of the disease is shown in the following case; the patient adjusted well to the disease, perhaps because she understood the nature of it.

CASE 5.—R. B., a graduate nurse, aged 58, was admitted to the psychiatric division of the Bellevue Hospital on Feb. 12, 1933, because she had been found unconscious on the floor of her room with the gas jets open on the day after the death of a sister with whom she had lived alone. She insisted that the episode was an accident, and that she had not intended to commit suicide. Her nephew (a physician) also did not believe that she had attempted suicide. At no time did she appear to be very depressed.

Examination.—It was known that she had had osteitis deformans for ten years. The disease affected the patient mainly through weakness of the back and diplopia due to proptosis of the orbital bones. There were also hypertension (blood pressure 170 systolic and 110 diastolic) and hypertensive cardiac disease.

She said, "My sister died two days ago. I swear that the gas had nothing to do with it. It was cold, and I got up to turn on the gas. I must have fallen

because of my weak back. I don't remember what happened. Now that my sister is gone and I am alone, I would like to stay in the hospital and get treatment."

When asked about her condition, she said, "I feel pretty good usually, although sometimes I see double. That has been for a year. In general, the Paget's disease doesn't bother me. I have accepted it—a bit too complacently, I think. We all have to die of something. I am stoic along every subject. I have Paget's disease, and I said to myself that I am not going to worry about it. I am very cheerful, and I don't become depressed easily. I don't know how I got the disease. I know it is rather an obscure condition. Does any one know how they get Paget's disease? I have no complaints to make."

Course.—Deep roentgen therapy was applied to the parathyroids, and calcium, including viosterol, was administered, with steady improvement. However, the treatment for Paget's disease will not be discussed here. It suffices to say that this patient has not shown any psychotic symptoms in response to a disease which, as an intelligent nurse, she understood as fully as any one can understand it, and which in her case is being treated as thoroughly as possible with promising results.

Comment.—Occasionally one observes psychoses associated with Paget's disease, in which the psychosis appears to be constitutional, as in the following case.

Case 6.—A. G. had been admitted to the psychiatric division of Bellevue Hospital seven times between the ages of 38 and 55 years, and had been transferred each time to a state hospital with a diagnosis of manic-depressive psychosis associated with paranoid ideas before Paget's disease was recognized or before there were any signs of diabetes. When the patient was 58 the diabetic condition was recognized and, in 1931, necessitated the amputation of a leg owing to gangrene. On June 6, 1932, the patient was readmitted to a surgical ward of Bellevue Hospital just after she had jumped out of a window and fractured the remaining leg. She was suffering from a recurrence of the psychosis. She said, "A girl threw me out of the window. She did it because my husband put her up to it. He always wants to get rid of me. I am too much trouble. I would be better dead." Similar ideas had characterized her previous psychotic episodes. The patient died on June 22.

Comment.—Similar cases have been reported, by Schiller,[10b] Smith[10c] and others, in which the psychoses occurred years before the Paget's disease was evident and also showed good remissions. One cannot resist emphasizing the constitutional defect in a person who has a manic-depressive psychosis, Paget's disease and diabetes mellitus, apparently independently. Paget,[9] in the first communication on this disease, called attention to the coincidence of malignant growths in persons with osteitis deformans.

COMMENT

The psychopathology of Paget's disease is due to a number of understandable causes. It is an obscure, mysterious disease which most people have neither heard of nor observed in other people. It is unintelligible. It progresses insidiously and slowly for many years, distorting the body form and structure in an unpleasant, ugly fashion, causing dull, obscure pains for which there is no relief, and distorting the special senses. In this way it attacks the postural model. Schilder,[2] in discussing the structure and psychopathology of the postural model,

showed that one's knowledge of his body is dependent on tactile and optic impressions and that the acquisition of this knowledge is a physiologic and psychologic experience invested with considerable narcissistic libido. As a body *Gestalt* it is dynamic and never static, but constantly changing, depending on deviations in its physiologic and psychologic structure, the emotional attitudes or the demands of the situation. Osteitis deformans tends to attack the physiologic structure of the postural model through both tactile and optic impressions, but the psychologic structure, emotional attitude and demands of the situational and intellectual grasp of the condition are such that the patient is unwilling or unable to accept or perceive the situation at its proper value. This is seen in a general way in cases in which the patient would not admit the obvious changes in body structure, and refused medical care and examination, viciously warding it off even at the cost of life; it is seen in a specific way in the patient who denied that the diseased, broken limb was his own but declared it was the "other fellow's," and in the blind woman who did not perceive her blindness.

The failure to perceive the disease and its effects is partly psychic but may also be partly organic. In more severe cases there are organic signs in the form of confusion, due either to an associated arteriosclerosis or possibly to effects of pressure on the brain. In case 3 blindness, at least, was organic. In a still more diffuse way this same mechanism may be seen in all the cases in which the patient did not accept responsibility for the disease, even when he recognized its presence, as he blamed it on outside persecution. Such delusional ideas are further fostered by the mysterious nature of the disease. Paranoid ideas are especially prone to occur when deafness is also present as a result of pressure. Deaf people are prone to feelings of suspicion and persecutory attitudes. In these cases vestibular disturbances further add to feelings of mysterious discomfort. In this disease pain is a particularly insidious symptom and leaves the patient powerless to defend himself against the unknown enemy. Pain is considered to be a sign of warning against danger. In this disease the danger is not evident, and the patient can in no way defend himself. This is a somatic disease that starts within the body but does not belong to the accepted concept of the postural model that is laid down in the central nervous system by the constitution and evolution of the personality and built up by the libidinous and social demands. It always remains foreign to the personality, both perceptionally and emotionally. Nevertheless, it sends impressions into the periphery of the personality through changes in the physiologic structure, sensations and pain. It also tends to thwart the patient's personal and social adjustments, either through the direct effects of the disease or by the ready tendency to fractures and injuries.

Two types of psychotic reactions occur. Paranoid pictures, more or less well organized, develop in connection with the feelings of mysterious changes and are especially liable to occur when the disease is associated with deafness. Emotional outbreaks are an expression of unwillingness to accept the distorting disease and its tendency to thwart personal drives. Some symptoms of imperception occur in all cases; they are related to the discrepancy between the body image and body structure. When it is possible for the patient to have some understanding of the disease and some hope from treatment, psychotic tendencies may be prevented or alleviated.

OSTEOGENESIS IMPERFECTA

Osteogenesis imperfecta was defined by Knaggs [7] as a disease characterized by a congenital defect in the osteoblast; it is recognized clinically by defective ossification of the cranium and by a multiplicity of fractures of the long bones from trivial causes. Patients with the fetal and infantile types usually do not survive. When it occurs later in childhood there may be no evident defect in the cranium, as in the case to be reported here. Multiple fractures of the long bones occur, with deformities of the limbs. The condition is also associated with a blue sclera, deafness due to otosclerosis, multiple sprains and a strong familial tendency. These features led Knaggs to the conclusion that the disease is a heredodegenerative disease, attacking the connective tissues. There is no involvement of the central nervous system, except as bony deformities may produce pressure on it. I have been unable to find any cases associated with psychoses reported in the literature.

CASE 7.—A. M., a single woman, of Italian extraction, aged 27, was brought to the Bellevue Hospital on Nov. 27, 1931, by a taxi driver who said that he had found her struggling along the street on crutches and crying. She asked him to take her to the hospital. She was several miles from her home in Brooklyn and said that she had walked, in an effort to reach the hospital, because "I had a quarrel with my mother and I could not remain at home. My mother does not like me. I was in Bellevue before, and I was treated kindly, and I decided to come back."

Examination.—The patient was short-limbed, undersized and unable to get about without crutches. The upper part of the body was well developed. The legs were shortened and deformed. There were scars from operations. The same was true of the arms, but in a less degree. The scleras were blue, and the patient was hard of hearing. There was some flattening of the vault of the cranium. The internal organs were normal. All the laboratory tests and neurologic examinations gave negative results. There was a mixed nerve and middle ear deafness. Roentgen examination of the skull gave negative results except for flattening of the vault. Roentgenograms of the long bones showed bowing of the lower extremities and retarded development of the articulations, characterized by exaggerated striation and extensive halisteresis. There was also decalcification of the pelvis and,

to some extent, of the vertebrae. In 1930, the patient had been in Bellevue Hospital for an operation to correct the bowing. A piece of the tibia had been examined microscopically and showed large narrow spaces lying in cloudy trabeculae. A diagnosis of osteogenesis imperfecta was established. At that time there was no evidence of any mental symptoms.

According to a sister, the family history revealed no other instance of osteogenesis imperfecta, disease of the bones, deafness or blueness of the sclera and showed no schizophrenia or other mental disease. The mother had an enlarged thyroid. The patient had appeared normal at birth and until she was a year old, when she was said to have had measles which retarded development so that she did not attempt to walk until she was 2. After that the legs began to break and broke "about a hundred times." Deafness began to develop some time later. The patient was with grandparents in Italy from the age of 6 to 15 years. While there she had operations on the legs. Since the age of 15, she had had no more fractures. The operation in Bellevue Hospital in 1930 did not help much. The sister said that since that operation "she has the ideas that all of us are against her. She says that her mother and father are to blame for her condition, because she is a cripple. She says her father is not her father. She gets angry at times and yells. She says that all the cars that go by the house are the doctors' from Bellevue, that they are coming around to look after her. She says that one of the doctors is her real father. She even says that one of the doctors is going to marry her."

The patient said: "My parents were ignorant and didn't care for me right when I was a child. They sent me to my grandmother in Italy, and it was worse there. She was even more ignorant. My brothers and sisters were all healthy. Last December I came to Bellevue, and they operated on me here. Every one was kind to me here. The doctors were kind, too. So when I went home I noticed that the doctors were always coming about the place. They would go by in cars and come in and switch on the light and switch it off again, and do everything to get my attention and show they were kind. Then there began to be talk about one of the doctors being my real father instead of my own father. I called my mother's attention to it, but she said it wasn't so and argued about it, and last night she got very angry, and we had a bad quarrel, and so I thought I would leave home and come back to Bellevue where the people like me and are kind to me."

Course.—Gradually the patient became suspicious and irritable in the hospital. In a few days she said, "Why don't you call that doctor who says he is my father and let me talk to him? No. I don't know his name, but you know who he is. I told the doctor who operated on me that my mother was guilty of an act with the doctor, and now he is trying to make it up with me. I wish they would tell the truth. I could stand it very well and forgive them. It was because of that that I was born weak and my bones were weak and broke, and they didn't give me proper care because they were ashamed. The doctor wasn't called every time, and they took me to the City Hospital when they could have afforded good doctors. Lately all these people go by in cars and nod at me and are kind. They are sorry for my sisters and ashamed for them because their mother did such a thing. But they are kind to me because I am weak. My family have noticed all these things, but they think it means that I have been having an affair with a man, which isn't true."

She continued to misinterpret the environment and to have ideas of reference while she was in the hospital. She would sit for hours at the windows watching the cars. She said, "They are coming around here, too. The brown car is the

car of some one of my people. They make so many sacrifices for me. That is what they did at home. There is a beautiful blue car. My father is in the brown car. I am not sure what he looks like. I don't see faces very well because they always turn around." (What sacrifices?) "He came after me when I came here. My people look upon me as a crazy person. That is the only way they will be saved. When I told them the doctors wanted to have an affair with me, they laughed and said, 'Nobody would bother you. You are too short, and there is nothing beautiful about you.' I know that. I am conscious of my state. I first noticed this thing long ago when my mother took me to the Bellevue clinic for my ears. The doctor looked at my mother as though he had known her for years. I tremble when I think of it. He operated on my tonsils. He always looked at her as though he recognized her. I think that doctor looked like me, and that made me suspicious. Many times I cross-examined my mother; she would get angry when I mentioned him."

The patient was transferred to the Manhattan State Hospital on December 9. While there, she discussed freely her delusions about the physicians following her and stated that her father was not her own. But on January 15 she was paroled to the parents and made a fair adjustment at home, although she was irritable at times and occasionally complained of some one following her.

Comment.—This is a case of osteogenesis imperfecta in an otherwise normal family. The patient suffered from "about a hundred" fractures during childhood, with resulting deformity. Schooling was interrupted, and social life was interfered with. She had no hope for marriage. She felt that she was scorned by her family. Gradually, as she reached maturity and faced the improbability of making an adult adjustment to life, and as physicians, to whom she looked for her only hope, failed to correct the defect of hearing and to straighten the crooked legs, she showed a tendency to identify them with herself and her family. She blamed the parents for her condition, at first simply by saying that they neglected her through ignorance and by failing to get the best physician and, gradually, by saying that this mysterious disease was the punishment visited on her because of the sin committed by her mother and the physician who looked like the patient and seemed to recognize the mother, and whom she believed to be her own father. Their neglect of her condition she regarded as the expression of their attempt to hide their shame. Her envy of her normal sisters she turned into shame for them because they had a mother who would commit such a sin; the same shame did not reflect on her, because she was already punished for it and was weak and pitiable. Not only was the one physician, whom she believed to be her father, kind to her, but they were all kind; they wanted to have an affair with her although her sisters said no one would bother with her because she was short and not at all beautiful.

The psychologic mechanism of this delusion system, with its identifications, ideas of reference, misinterpretations and auditory hallucinations, is pathetically obvious. It has many of the characteristics of the paranoid schizophrenic system; however, there was no dissociation in the emotional response, and there were no catatonic or other physiologic evidences of schizophrenia and no evidence of deterioration. Such delusional systems are not uncommon in persons suffering with deafness alone. In this case it is an attempt to explain a mysterious disease which has been deforming the body for many years and which has been

destructive to the chances of making an adult adjustment, and has thwarted the normal instincts and ambitions. In some way also it affords a partially satisfactory fantasy life.

One is reminded of the organ inferiority theory of Adler,[11] which is based on the theory that diseases attack organs that are inferior by heredity, and that these inferior organs may be responsible for the feeling of psychic inferiority which becomes the foundation for neuroses and psychoses. Further, he believed that, because of the hereditary nature of the organ inferiority, there is always an accompanying inferiority in the sex apparatus. One does not feel in this case, however, that there is necessarily any inferiority in the sex apparatus, but rather that the normal sex drive is thwarted by the limitations of the distorted body and that the delusional system arises from the unusual, mysterious nature of the disease, the feeling that it is not a part of the body image and therefore that some one else must be responsible for it; since it arose in childhood, obviously the parents must be to blame.

DWARFISM

Dwarfism is not organically a unitary condition. In a general way it may be said to be the result of some disturbance or retardation in growth. There are a number of different types of dwarfs [12] dependent on whether the growth interference is a general unknown factor which results in the proportionate or true dwarf, who is merely a smaller than average person but who shows no evidence of infantilism, or whether it is one of the specific or nonspecific endocrinal or osteogenic disturbances that occur in cretinism, polyglandular dwarfism or achondroplasia. Some disturbances of growth may also interfere with the development of the brain, with subsequent retardation in intellectual or emotional development. This is especially true of the endocrine disturbances in which infantilism is associated with the dwarfism. This is not the condition of interest in this study, but rather the question of more or less acute psychoses that develop in adults in reaction to dwarfism. Dwarfs have often been held in high esteem; as a result many have lived happy and useful lives. In a book on "The Mystery and Lore of Monsters with Accounts of Some Giants, Dwarfs and Prodigies," Thompson [13] gave an account of the part that dwarfs have

11. Adler, Alfred: Studies of Organ Inferiority and Its Psychical Compensations, translated by Smith Ely Jelliffe, Monograph 24, New York, Nervous and Mental Disease Publishing Company, 1917; Individual Psychology in Psychologies of 1930, Worcester, Mass., Clark University Press, 1930, p. 395.

12. Bassoe, Peter: Dwarfism, in Barker, L. F. A.: Endocrinology and Metabolism, New York, D. Appleton and Company, 1922, vol. 2.

13. Thompson: The Mystery and Lore of Monsters and Accounts of Some Giants, Dwarfs and Prodigies, London, William and Norgate, 1930.

played in the court life of many countries, as jesters, favorites of the court ladies and court advisers. They were much sought after and held enviable positions in Egyptian and Roman courts, and in all European courts during the Middle Ages and until recent times. The most recent of famous dwarfs was the American prodigy, Tom Thumb. Aside from being appreciated by the world at large, dwarfs were collected together and were accustomed to seeing others like themselves; they did not consider themselves to be unusual or undesirable persons. In present times some dwarfs have a fairly good life as professional show people and are allowed to capitalize, not only financially but in general appreciation and satisfaction of libido, on their defect. Such persons make good adjustments and do not appear in psychiatric clinics. The most sympathetic description of the psychology of the dwarf is written in the form of fiction in Walter de La Mare's "Memoirs of a Midget."[14] The author, in autobiographic style commented on the problem which I am presenting as the basis for the psychoses which occur in such persons.

"How many times, too, have I vainly speculated what inward difference being a human creature of my dimension really makes. What is—deep, deep in, at variance between Man and Midget?" (p. 15). . . . "How secure and perhaps wholesome is Man's self-satisfaction. To what ideal does he compare himself but to a perfected abstraction of his own image? Even his Venus and Apollo are mere flattering abstractions of his own he- or she-shape. And what of his anthropomorphic soul?" (p. 49). . . . Perhaps this "inward difference" "between Man and Midget" is that the personality of the midget does not accept the postural model which is not "a perfected abstraction of man's own image." Even though the midget of de La Mare's fantasy says, "It is true that my body is one of the smaller works of God" (p. 21) and thereby tries to consider it of some specially precious significance at least to God; still in her dreams which best express her unconscious concepts, she states: "I dreamed that I was walking in a strange garden—an orchard. And as it seemed I was either of common size or this was a world wherein of human beings I was myself of usual size" (p. 58).

I shall present two cases: one of an atypical cretinoid dwarf of normal intelligence and personality until the age of 27 years, and one of a proportionate or true dwarf. By way of contrast, two other cases of dwarfism without psychoses will be mentioned: one of an achondroplastic dwarf, and another of a polyglandular dwarf.

CASE 8.—F. C., a cretinoid dwarf, of Russian Jewish extraction, aged 27, was brought to the psychiatric division of Bellevue Hospital on Nov. 27, 1931, by her mother who said: "She was always nice until three or four months ago. Now she is suspicious of every one. She believes we all want to harm her, and stays in bed all the time and screams if we want to touch her. She believes we want her to die. We cannot take care of her at home any more for this reason."

14. de La Mare, Walter: Memoirs of a Midget, New York, Grosset and Dunlap, 1922.

The family history was irrelevant. The patient was apparently normal at birth, and was the second of two children. Nothing unusual was noted until she was 2 years of age, when the mother took her to a physician for a cold; he told the mother that something was wrong and gave her pills which apparently were extract of thyroid gland. The child took these pills "to grow" until she was 6; then her hair began to fall out, and the pills were given in half strength. At 15 years the pills were stopped. The patient menstruated a few times at the age of 16, but the menses ceased and did not return. She was taken to many clinics but was told that nothing could be done to help her. She went to school until she reached the seventh grade, when she stopped because the children teased her. She helped her mother in a small shop. Until recently she had been happy and contented at home and was well liked by relatives. In the past few months she had had the idea that the family wanted to kill her, and she was afraid of them.

Physical Examination.—The height was 3 feet (91.44 cm.). Most of the features were typical of a cretin, except that she had thick, fine black hair. There were also myxedematous features: The bridge of the nose was flat, the forehead broad, the lips and tongue were thick and the neck was short. An epicanthus was present; otherwise the eyes were normal. Speech was mumbled. The legs were bowed and the feet flat. The abdomen was protruding. Normal breast tissue was present. The teeth were poor and widely spaced, and there was a high palate. The skin was rough, dry and myxedematous. The thyroid gland was not palpable. The heart and lungs were normal. The reflexes were normal. Roentgenograms showed a dolichocephalic skull and a normal sella turcica. The long bones were short and stunted. The epiphyses showed incomplete synostosis. The carpal scaphoids were fissured and fragmented.

Course.—On December 10 the patient was given 2 grains (0.13 Gm.) of thyroid extract. Steady improvement occurred in the general condition until December 28, when the pulse rate became elevated to 105 and her hair began to fall; the dosage was then reduced to 1 grain (0.065 Gm.) of thyroid extract daily.

Psychometric examination on admission gave: with the Terman scale, a mental age of 10 years and 2 months (intelligence quotient of 73 on the 14-year basis); with the Otis self-administering scale, an intelligence quotient of 76, and with the Porteus maze test, a mental age of 7 years. A month and a half later, after the patient had responded to thyroid treatment, the Army alpha scale gave a mental age of 12 years and 9 months (intelligence quotient, 91), which appeared to indicate that she was not defective.

On admission the patient was fearful, reticent and evasive, somewhat dull and preoccupied. She said, "People bother me. They laugh at me. They make a laughing-stock of me. The neighbors spoke behind my back. They said bad names. They called me midget and all that. I didn't hear them say it, but it came out in some manner. Sometimes they put their spirits on me. It is this cursed generation. Lately they put the curse upon the water. I don't know who did it. Just lately, when the water started to come out, the spirits began to talk. Before, the water wasn't cursed. The spirits say, 'How is the F—king business.' I don't like to tell everything they say. The spirits say, 'Coming' and 'Not coming.' The spirit speaks itself. The spirit tells me jokes. I can't remember them."

On the next day she told the following story of her life: "I went to school till I was 16. I was in 7B. They always let me out of school fifteen minutes early because I couldn't get down the stairs as fast as the others. One boy went down two steps at a time. I'd roll down if I tried to do that (laugh). I played with the other children. Some were kind and some weren't. After I left school

I stayed home. I was happy until they started to mistreat me. I had trances when they didn't treat me well. That is since I got sick with asthma. A few months ago I got nervous. It is my aunt that is to blame. She is jealous of me because I was the pride of the family and because I was good and behaved myself and was loyal and considerate. She has one child. I like her very much, but she was not taught to pray. I used to pray, but I lost my faith in God. I don't want to say why."

The patient was obviously proud of her hair, and when this was praised she said, "Yes, it is pretty, but my aunt put a curse on it. It isn't so pretty any more. It was curlier and long, in two braids. She was jealous and put a wish on it. Now it is thinner and nearly as straight as hers, and they had to cut it short. That aunt used to be kind to me. She gave me gifts, wrist watches and everything, but she turned against me because I was so good. They say that when I was young my aunt pushed me over and dropped me or something, and that is why I did not grow. Lately, when I am in a happy trance, it seems as though I am as big as other people. I had noticed that I had grown 2 inches (5.08 cm.) in the last two months. The family didn't notice it until I told them. I noticed that I could walk faster when I was in the park. Some people say that you are 2 inches taller in the morning than you are at night. Yes, I have noticed that, too."

Three weeks later she was showing some signs of improvement as a result of treatment. She was more amiable and talked more freely. It was noted, however, that she often hid her head in the bedside table and talked or cried to herself. Sometimes she laughed and said a nice big man was talking to her. It was also noted that she had taken the hem out of her dress in order to lengthen it. When questioned about these matters she said, "Sometimes I have trances, mostly at night. Before, I used to have them a lot. I would cry, and sometimes I would talk. I had one like that a few days ago. I think men are talking to me. Yes, I rather like it. I feel that I have grown up a few inches since I am taking the pills. I can tell it in my appearance. It shows up in my height. That is the reason I let down my hem. I did it a few days ago. I grew some before I came here. I could tell by the size of my thighs. They got bigger. I had one nice trance. I thought I had a carriage with a doll. Some of the trances are good and some are bad. I am feeling better now. I felt dopey and confused before I started taking the medicine."

She continued to improve, was happy and cheerful in the ward and made many friends until about three weeks later, when she contracted a cold, became toxic and showed a recurrence of the former emotional suspicious state. She said, "My father is dead; I know he is; otherwise he would come to see me. My aunt told me the radio was out of order. If my father was alive it would not be out of order because he would fix it." However, when she recovered from the cold she recovered from this emotional episode and regained insight into her mistaken ideas. She continued to improve physically and mentally, so that in February she showed no signs of myxedema, although the cretinoid features persisted; she was able to pass the mental tests at the higher level and made a good adjustment to the ward. However, she continued to be a difficult type of person, who demanded and enjoyed a good deal of attention but became jealous and sulky if any one else received attention. She was not anxious to return home, and to have her return there did not seem wise because she could not compete with her growing normal brothers and cousins. She showed a special appreciation for masculine attention and did what she could to win the affection of any attentive interns.

On February 1 she was transferred to Central Islip State Hospital. It was reported from there that she made a good adjustment and got along well in the social life of the hospital, though her mood was variable and at times she was petulant. She was evasive about her delusional ideas, but in June, five months after admission there, she admitted auditory experiences which she said came from her soul, but she showed no evidence of persecutory ideas and acknowledged that she was happy in the hospital. She was transferred to the Pilgrim State Hospital, where she is at the present time, and it is reported that she has been disturbed for some time. She has paranoid ideas against every one with whom she comes in contact. She imagines that attendants abuse her, ridicule her small size and assault her sexually. She admits hearing voices of men and women who call her "midget" and "shrimp," and she speaks of having trances at night. She will not discuss her ideas with the physicians because, she says, they will "condemn" her. She is said to be resentful and irritable, uncooperative and resistive. She is receiving no thyroid medication at present, as she refuses it, saying it is poisonous.

Comment.—This cretinoid dwarf was treated a great part of her life and reached a fair intellectual and emotional maturity. She made a good adjustment so long as she was a favored child and could adjust as a child. As her sibling and cousins began to make adult adjustments and she herself reached an age when she could no longer be considered a child, she could not fit the rôle of a woman who should be considering marriage. A psychosis was precipitated during a period of poor health, in which myxedematous features appeared. The patient became fearful and suspicious of relatives, believing that they wished to be rid of her. There appeared a rather elaborate and well organized delusional system to account for the dwarfism, on the basis of a jealous aunt who had a normal, healthy daughter of whom the patient was jealous. She said that the aunt had dropped her when she was small, so she could not grow, that the aunt put a curse on the water to throw her into trances and that, out of jealousy, the aunt made her beautiful hair (her most normal feature) fall out.

Thompson [13] quoted an old Saxon tradition by Delrio to the effect that "Parents greedy of gaine, very wickedly, with certaine medicaments cause their children's growth to be stunted that they prove dwarfs." In a somewhat similar way, the patient in case 7, with osteogenesis imperfecta, blamed her parents, while in various ways the patients suffering from Paget's disease explained the mystery of the origin of their disease. De La Mare's midget also said: "At times I have been tempted to blame my parents for my shortcomings."

The patient's trances were of two kinds: unpleasant ones (for which she blamed her aunt) in which she was nagged by unpleasant sex hallucinations, and pleasant ones when the fantasies gave way to the postural model that fitted her more deeply embedded concepts and she found herself of normal size, or at least she noticed that she was getting larger, her stride was greater when she walked and she "felt" bigger. At times she deliberately hid her head on the bedside table and played with fantasies that she was being courted by a man of normal size. In the ward she found it necessary to lengthen her skirts. She showed signs of definite improvement under treatment with thyroid extract. As the myxedematous symptoms improved, the intellectual grasp and emotional stability improved, and she gained partial insight into the psychotic delusions, but she never seemed stable enough to return home and compete with normal adults. For long periods she made good social adjustment in hospitals, but subsequently she returned to the delusional state.

CASE 9.—E. L., a proportionate male dwarf, 3 feet 11 inches (109.22 cm.) tall, aged 32, weighed 75 pounds (34 Kg.) and was well proportioned, with well

developed, though small, primary sex organs and well developed secondary sex characteristics except for a scanty beard which he shaved twice weekly. The facial features appeared somewhat prematurely aged, and the skin was dry and parchment-like. The basal metabolic rate was minus 10. Otherwise physical, laboratory and roentgen studies gave normal results. Intelligence was good. With the army alpha scale the intelligence quotient was 105. The family history was free from any abnormalities. The father and four brothers were all about 6 feet (182.88 cm.) tall. Three sisters were about 5 feet, 6 inches (167.64 cm.) tall. One sister and the mother were about 5 feet, 2 inches (157.48 cm.) tall. The patient had appeared normal at birth, and it was first noticed when he was 3 years old that he was not growing as he should. At the age of 6 years he was sensitive about his height and he would not attend school until he was 9. He did good work in school and graduated at 14. He had several offers to join circuses and shows, but he was very sensitive about his condition and refused. After leaving school he practically hid himself away on a farm. He was devoted to his youngest brother and associated closely with him; after the brother married, about five years ago, the patient became more and more reticent and seclusive. He had no associates and made no friends. He lived with his mother, sister and brother-in-law and became progressively suspicious of them.

On March 3, 1932, he was arrested on a charge of possessing a revolver and was sent to the observation ward of Kings County Hospital. His sister said that he had been nervous for some time, that he had imaginations and had said that he was the Son of God and was more powerful than any of them. He believed that they wanted to do him harm. However, at the hospital he was evasive and said: "I bought the gun for no real reason at all. Me and my brother were thinking about going on a trip, and I wanted the gun for that. I had the gun for protection. That is why I was arrested. I have been worried some. I was restless and couldn't sleep. At home they wanted me to see a doctor. That made me worry. They had an idea I was nervous. I know it is a frame-up, but it is nobody's business but my own. I thought they were friends. It was on account of the frame-up that I had the gun. If I tell you about it, it will cause more trouble."

He was declared insane; the charge was dropped, and he was committed to the custody of his sister. However, he did not get along well at home and moved about from one relative to another or stayed in the attic of his mother's house where she had to carry him food. He complained that various members of the family were jealous of him and wanted to steal his property. The property he referred to was not his own; it was a house and lot that belonged to his mother and in which his sister and brother-in-law also lived. He was especially suspicious of the brother-in-law. He was brought to the psychiatric division of Bellevue Hospital on Aug. 7, 1932, at the request of the mother.

Mental State.—The patient said: "They don't treat me right. They want to put me out of the way. They want my money and my property. It is hard to say what the trouble is. I want to find out. It is much about nothing. I can't figure it out. It might be jealousy or spite work. I have some property—almost an acre with a house on it. I earned the money to buy it by farming. I live upstairs, and I rented the downstairs to my mother. She lives there with those people (sister and brother-in-law). I stay up there alone and read and write. My mother brings me food occasionally. It is my brother-in-law that has it in for me. He won't speak to me sometimes. He might think I have a little money. He probably thinks he can get me out of the way. There is something wrong

with the way he acts. I try to keep out of his way. They have all tried to buffalo me. I can't talk about it. If I tell you about it, it will be against me. I never can make a move. Whenever I try to go into business, they hold me back."

When an attempt was made to discuss his size he was also evasive and uncommunicative, and showed a definite tendency to minimize the defect and its influence on his life. He said, "I don't know why I am small, and I don't care. That is past. There are many people in my family that are small. My mother's sister and my grandfather were small, though not smaller than average. One sister and my mother are almost as small as I am. I think they are about 4 feet, 3 inches (129.54 cm.)." (This was not true—they were 5 feet, 2 inches.) "I never noticed that I was especially small. In school I was about the size of the other boys, as I remember. Well, perhaps when I graduated I was smaller. It didn't matter. I could do everything in farm work except plow. Sure I got out with girls whenever I wanted to and got along with them all right. I have been thinking of getting married, but don't have any one in mind just now. I have had plenty of chances to enter the movies but I didn't want to."

Course.—The patient was transferred to Central Islip State Hospital in August 1932, where he still remains. He continued to be evasive and uncommunicative, still believing that his brother-in-law "framed him" through spite or jealousy. However, he seemed satisfied and contented in the hospital, although he appeared dull and uninterested. At no time was there any evidence of hallucinations, bizarre behavior or catatonic features.

Comment.—This dwarf, of good intelligence, was an isolated case in his family and within his own experience. He refused to consider capitalizing his defect by entering a circus or show and became more and more seclusive. He tended to minimize his defect and to show retrospective falsification as to his defect as a child. He claimed that he was making friends among normal women and contemplated marriage. Apparently he had had some grandiose ideas at one time, believing himself the Son of God and all-powerful, but he corrected these and secured a gun to protect himself against relatives who, he believed, wished to steal from him the property which he possessed only in his delusions. He made a fair but dull adjustment in the state hospital. This man could not accept his small stature and tried several methods of adjustment: grandiose delusions, trying to add a cubit to his height by means of a gun, appropriating property that did not belong to him, and, finally, imagining himself the object of jealousy on the part of others about him, especially the husband of his favorite sister. To himself and others he denied the defect.

Case 10.—M. M., a polyglandular dwarf, aged 19, 34 inches (86.36 cm.) high, and 29 pounds (13.2 Kg.) in weight, was brought to the clinic for mental hygiene of Bellevue Hospital by his mother because of recently developing convulsions. He had been on exhibition since the age of 8 years and was a successful showman at the time. He had the intelligence of a moron, with some special abilities. The family history was entirely without significance. He was a full term baby, with normal birth, who weighed 9 pounds (4.1 Kg.) at birth according to the mother. He was the fifth of six children.

The patient displayed a happy, extrovert personality, with infantile features. The mother said: "He is always active, always likes to be doing something. He takes part in regular shows and movies and likes it very much. He likes to do everything everybody else does. He plays cards and checkers very well. He wants to play golf. He never worries or fusses about the fact that he is small. He seems happy all the time, always smiling and congenial."

Comment.—This patient made a good adjustment with his infantile personality and simple level of intelligence, except for the convulsions. He made good contacts with others of his own type. He had no problems. Sexually he was underdeveloped and was not presented with the problem of normal adult heterosexual adjustment, which caused great difficulties in the patients in cases 8 and 9. However, this patient was younger than the other two patients were when they became psychotic.

CASE 11.—S. A., an achondroplastic dwarf, who was brought to the psychiatric division of Bellevue Hospital on Aug. 15, 1932, at the age of 90 years, had lived a normal and happy life until that age, when cerebral thrombosis resulted in aphasia. She outlived the rest of her family, who had lavished on her every blessing they could find to make her happy. She had private tutors in youth and had been trained to sing and encouraged to enjoy herself as much as possible. She had been treasured and humored throughout her long life and had never had a psychotic episode.

Summary.—I have presented two cases of psychoses in dwarfs, in which the content of the delusions has shown clearly the conflict between the patient's inherent concept of what the body image should be and what it actually was. There are clear efforts to deny the small body form. The struggle becomes paramount when the interests of adult life are thwarted. Such persons adjust so long as they are favored children in the household and are also children in their own personalities. But when the demands of normal growth to adult life for social, industrial and heterosexual adjustments cannot be met, they seek an explanation in psychoses that are characterized by emotional disturbances and delusions that place the blame for the condition on those who are responsible for their existence, and in delusions that tend to deny the defect. For periods of time they make adjustments in state hospitals. The libido seems to be created on the plan of a normal body image, but the means for satisfying it is limited by the diminutive physical body; thus there arise the discrepancies between strivings and ability for accomplishment. When the strivings are reduced to the size of the body by an infantile emotional and intellectual level, or when the means of satisfying them has been adequately met by a devoted family and an admiring social regimen or by association with others of their kind, psychoses may be avoided.

In terms of de La Mare's midget, the discrepancy is "that variance between Man and Midget" created by man's "perfected abstraction of his own image" which, in some way, is "the Anthropomorphic Soul," and if the body does not fit the soul, the discrepancy may lead to a struggle that can find no solution in this world of reality and leads to a splitting of the personality into a schizophrenia-like psychosis.

DISEASES CAUSING IMPAIRMENT OF MOTOR AND SENSORY FUNCTION

So far, I have considered diseases that distort the bony framework and those that disturb the normal growth of the body. I shall now con-

sider cases in which the insult to the physical personality is also a major and disfiguring one that works through the nervous system. These diseases disturb mass parts of the body by disturbances in motor functions and also in some cases peripheral sensory functions. Such diseases represent a serious injury to the concept of the postural model or the body *Gestalt*. The study includes single cases of Little's disease, poliomyelitis and pernicious anemia with subacute sclerosis.

CASE 12.—E. B., an unmarried woman, aged 40, presented a case of Little's disease due probably to an injury sustained at birth. On Jan. 20, 1932, she was brought from a theological seminary to the psychiatric division of Bellevue Hospital with the complaint that she had shown acute mental symptoms during the morning chapel services. She threw her shoes at the presiding attendant and later, when taken to the chaplain's office, broke a window. She claimed that she was a victim of poisoned food, which members of the seminary were using on her for experimental purposes.

Examination.—Physically, the patient was poorly developed and poorly nourished, frail and undersized, with spasticity in all four limbs, more marked in the legs than in the arms, and more marked on the left side than on the right. On the left side there were also muscular atrophy and intermittent tremors of both the arm and the leg. The left leg was shorter than the right, and there was scarring as evidence of previous operations on the left hip.

The family history was without significance. The patient was a full-term child, born by breech presentation, and faulty extraction was said to have resulted in paralysis of the left side. At the age of 8 she had a severe attack of whooping cough. At the age of 10 she fractured her nose; this necessitated an operation at 18. At 16 she had a tenotomy operation done to alleviate the difficulty in walking.

She went to school at 6 and was a bright pupil. She was promoted regularly and completed two years at Elmira College. Afterward, she attempted to work in an office for six months, but found herself too nervous to succeed. She also attempted to run a greenhouse, but gave that up also. She became increasingly restless and dissatisfied with her lot until she was admitted to the Binghamton State Hospital in February 1922, at the age of 30. The analysis of her make-up and the psychosis at that hospital is significant.

"As a child she was considered of normal make-up. She had many boy friends but no love affairs. She was always very fond of her father but inclined to blame her mother for her physical condition. In college she had a great ambition to become a writer but later realized, with considerable shock, that she had not the required ability. . . . She was greatly impressed with religious ceremony. . . . It was felt that her attitude toward sex was normal but that she had reconciled herself to a life of spinsterhood because of her physical disability. . . . She was especially fond of older women.

"The psychosis was believed to have begun six years before admission, when she became depressed and restless and on one occasion wired her mother in the middle of the night to come at once because she felt she was going to be very sick. She believed that two of her women friends wanted to adopt her to take the place of her mother, and these women had to treat her very tactfully to avoid hurting her feelings. . . . She began to show definite antagonism to her mother and avoided her company as much as possible. On several occasions she said she

heard her neighbors call her names and, while she could not see them, she felt their presence and saw their shadows. About six weeks before her admission, she warned one of her girl friends to stay away from her or she would shoot her. One night she put on her coat over her nightdress and went to the river, but she returned and said her pet cat called her back. She visited her sister's home and expressed a fear of being left alone with the children and pets, as she thought she would be blamed if anything happened to them. She became more and more suspicious and said people talked in opprobrious terms of her physique and morals.

"On admission to the hospital in September 1922, she said that she could not trust herself and that people avoided her and looked at her as though she were a ghost. On the ward she maintained an attitude of superiority with irritability. She exhibited ambivalent erotic tendencies. At times she was attracted to female physicians, and at other times to male doctors. She believed that a nurse who gave her sex information in her childhood had spread stories to the effect that she was a pervert. She admitted sadistic impulses toward children and animals. . . . Her formulations were traceable to basic sex difficulties and her sense of inferiority, for which she compensated by profession of intellectual and physical prowess. After three months she improved and was paroled on Feb. 24, 1923."

After discharge she did not get along with her family and would not live at home, especially because she distrusted her mother. She had affiliated herself with the theological seminary, where she attended the lectures and religious services.

Examination.—After admission to the psychiatric division of Bellevue Hospital she said: "I was brought here by the police. I interrupted a church meeting. I have a trembling in my arm, and I believe they are experimenting with me and trying to cure it. It makes me shake all over, and the only way I can relieve that is to jump up and down. They figure that if they knew how to cure the trembling in my arm they would know how to cure others. It is an experiment. They feed me medicine and then feed me food to feed the medicine. It seems to influence my sex organs. It is not normal. I have waking dreams that I am loved. I have been studying in a seminary where they make ministers. I think they would like very much to learn how to help people. I am afraid it will hurry along the time when I can't have children. If some one would fall in love with me, I would like to have a baby. The ministers do not wish to hurry the time I will not have babies. They do not know that they make this feeling. It is ignorance on their part. They come in my dreams. It is an illusion. It is pleasant if it is a nice person. But there is a colored man that I simply can't stand. There are three white men. I can tell them apart because their ways are different. There is one I enjoy the most. I feel like I am flying, to be with him. There is candy and fruit. It never comes to the point of sex relations. I feel his arm. I feel that he started and withdrew. I know all the time it isn't true. I never see him. He has very broad shoulders that I enjoy very much. The second man is younger. I can't tell you how I know. He sings. He can sing, and I feel that I can sing. I have no voice, as a matter of fact. All the time we are together we sing hymns together in my mind. It never comes to the point with him either. The third one—he really comes to the point. I get complete satisfaction with him. I know him in life, and I don't like him. There may be two black men, I am afraid. Or there may be only one. It never comes to satisfaction—I know and interfere—I get up or something. I know he is colored because I smell him. I think it is his own breath I smell. I know these are all illusions,

because there is nobody there. But I can feel their hands on my hair and face. I can feel their breath. Sometimes there are little hands that touch me; the hands of children.

"When I have these fantasy dreams, I always feel normal myself. I am myself but my limbs are normal. I've always had fantasies like this, but now they seem real, in the last six months. It is because I won't let them be real. I've never even been kissed. No one ever wanted to. I was brought up to think that thoughts like this are wicked. Therefore, I think something is wrong, and they must be poisoning me with some experiment. I always enjoy my menstrual periods. At such times I am more alive to beauty, to the sweetness of life. I am afraid the medicine they are giving me will stop them, and I don't want that.

"My lameness has never really bothered me at all. It is just as if it wasn't. I've never minded. I have danced and played all sorts of games."

The patient was transferred to Rockland State Hospital on January 27.

Comment.—In this case, an injury sustained at birth had severely distorted and crippled the body, and the patient struggled against the conflict between her body as she found it and her constitutionally and socially determined concept of what the body should be, especially with the sexual problems of adult life. She began by blaming her mother and gradually turned more and more against her. First, she turned to other women as substitutes, and then turned away from them too. Emotional instability was more marked around the menstrual periods. In a first psychotic episode she expressed a fear of injuring children, while in the second she expressed fear that she will never have children. As a religious person she sought companionship in a seminary where "they make ministers," and fantasied love affairs with them until the fantasies seemed nearly true to her; then she blamed them for experimenting on her. The tremor of the spastic limbs became a part of the sex fantasy. In the fantasies she acquired the normal postural model—her own normal postural model. But even in her real life, she said her lameness "is as if it wasn't." In this case one sees the effort to explain the discrepancy between the body and the concept of the ideal postural model by mistreatment from the mother; she tried to deny its existence, and in her struggle with thwarted adult libido drives showed marked emotional difficulties with sex fantasies that at times became delusional.

A similar problem is seen in the following case of poliomyelitis, although this woman has been able to make a better adjustment to life than the last one, with only episodes of emotional disturbance as a result of thwarted instinctive drives. The emotional episodes have not as yet become delusional.

Case 13.—E. H., a single woman, aged 36, at the age of 5 had suffered an attack of poliomyelitis which had paralyzed the back and lower limbs. The arms were not affected. There was a severe scoliosis of the back, with deformity of the chest and defective development, weakness and atrophy of the legs. The patient had had thirteen operations on the legs. She had defied her family's prophecy that she could do nothing and had completed a high school and a business course, had a public stenographer's office, and a car which she drove with special attachments, and earned her own living.

She was brought to the psychiatric division of Bellevue Hospital on Oct. 28, 1933, in a profoundly dejected and toxic state, by a man friend who said, "I had not seen her in three months and I went to see her and found her in a nervous hysterical state. She told me of a love affair that had broken up. After I left her, I phoned her several times, and as I did not get an answer, I went back, broke into her office and found her all in a heap. I thought she was dead."

She admitted later that she had taken a number of tablets of a barbital derivative with amidopyrine. After admission she sobbed dramatically and repelled all attention and food, insisting that she must die. Between sobs she told her story: "Infantile paralysis at 5; thirteen operations. Family said I couldn't do anything. Wanted to earn my own living. Typing and stenography. Pride and will power. Made good. Got my own public office. Worked so hard. Just paid off all my debts. Could die now—all debts paid. Thought I was like other people—someone would love me. Thought I could give something. Thought I had something to give. Nobody's fault but my own—my mistake. But now I feel that I have no worth—no value in life. I am so tired now that I can't come back. I want to die. I have a right to decide." Two days later, when she had improved, she said, "Somebody must have brought me here; I don't know who. I didn't want to live. I don't think any one else knew it. It just didn't work, what I wanted to do with my life. It was just too much. Nobody can help me. The trouble is I am no fool; I have reasoned it all out."

Her sister said, "She is the strongest character in the family. We thought she couldn't do anything. She has done more than the rest of us. She has this office and her car. She always has a bunch of stalwart young fellows around, who are supposed to be working for her—helping her with the car and this and that. Actually they 'do' her; they take advantage of her, they drive the car and hang around her office. Every one else knows there is nothing in it, but she likes to think it means something. She likes to think they are 'beaus.' She has got upset like this several times, but never so bad."

Course.—After ten days the patient had entirely recovered from the emotional upset and was discharged to her family with renewed courage.

Comment.—In this case the behavior was nearly all at a conscious level. The patient refused to accept her handicap as such and maneuvered to do everything that her sisters did. Most of these things she had accomplished, but satisfaction of her adult heterosexual drives failed her. This led to episodic emotional disturbances.

In considering a case of psychosis with pernicious anemia, I do not mean to intimate that all psychoses associated with pernicious anemia and subacute combined sclerosis can be explained by the mechanisms suggested here. Hackfield [15] reported seven cases of psychosis associated with pernicious anemia and contended that the pernicious anemia bears no direct etiologic relationship to the production of the psychosis but that the psychosis is a separate entity. These conclusions do not seem satisfactory and have not been the opinion of others. Woltman [16] reported six cases in which he emphasized the importance of the anemia as a cause of psychosis, and Barrett [17] summed up the psychotic picture

15. Hackfield, A. W.: Studies of the Etiological Relationship Between the Somatic and Psychotic Disturbances in Pernicious Anemia, J. Nerv. & Ment. Dis. **76**:31, 1932.

16. Woltman, H. W.: The Mental Changes Associated with Pernicious Anemia, Am. J. Psychiat. **3**:435, 1924.

17. Barrett, A. M.: Mental Disorders Associated with Pernicious Anemia, Fifth Biennial Report State Psychopathic Hospital, University of Michigan, 1916; quoted by Woltman.[16]

by saying that irritability and suspiciousness form the groundwork for delusions of persecution, the content of which is usually influenced by the somatoneurologic findings. It is this last point that I wish to emphasize in reporting the following case.

CASE 14.—R. D., a married, childless woman, aged 50, who was born in Germany and had come to this country ten years previous to this history, was brought to the psychiatric division of Bellevue Hospital on Aug. 6, 1931, from a police station to which she had gone complaining that people threw acid in her mouth and talked about her at night. She believed that this had been instigated by her husband from whom she had been separated since 1922. She stated that she had had pernicious anemia since 1925; at that time she was treated in the City Hospital with two blood transfusions; since then she had received a diet with liver and liver extract in several clinics. Weakness of the legs, burning of the tongue and tingling of the hands were first noted two years before the present admission.

Examination.—The erythrocyte count was 3,880,000, with 70 per cent hemoglobin and a leukocyte count of 5,950. Poikilocytes and macrocytes were present. The mucous membranes were pallid. The tongue was smooth, beefy and atrophic. The knee and ankle jerks were absent, and vibratory sense and sense of position were diminished in the lower limbs.

She said, "I have been sick with pernicious anemia since 1925. I had blood transfusions and lots of liver and liver extract. I have not lived with my husband for nine years—since 1922. I don't know whether we are divorced or not. I think he is responsible for this condition. He threatened me. He made me go to the medical center for a puncture on my back. I am sure he was back of it—through some people at a funeral of a woman at our church who died. Since then my legs have been bad. He has people follow me around a good bit. He comes to my house in disguise. I can tell him by a scar along the jaw bone. He is responsible for my condition. For months they have thrown stuff around like acid. I have to sleep with a wet handkerchief on my face. I couldn't eat food I had not cooked in the house. If I cooked liver in the frying-pan it would swell up like a bubble. Some of them have been doing me dirt, putting acid on my back, and it smells awful. All around my clothes you could smell it. I would take a drink in the morning, and the next day my skin would be as yellow as saffron. The day I went to the police I was sitting in the movie and some one threw acid in my mouth. They squirted something that got into my mouth and stomach and lungs. It made me vomit. The first times they did it was after Christmas. It felt like creosote, and my bowels didn't move for five days. Sometimes it smells like old potato skins. The acid burns my nose, tongue, mouth and throat. People say things about me among themselves. If I ask them anything, I get a dirty look. People talked very nasty about me."

Course.—The patient was transferred to the medical service of Bellevue Hospital for treatment, which resulted in a good remission of the blood picture, after a month, with an erythrocyte count of 4,200,000 and hemoglobin, 85 per cent. However, there was no improvement in the neurologic signs, and the delusions persisted essentially as before. She was transferred to the Manhattan State Hospital.

Comment.—This patient had severe and, to her, mysterious changes in the nervous system which resulted in difficulties in walking, numbness in the hands associated with burning of the mucous membranes and certain visceral crises, which the patient accounted for in their entirety by persecutions caused by her husband, who deserted her several years before the onset of the illness.

SUMMARY AND CONCLUSIONS

This study has presented cases of psychosis associated with somatic disease producing a severe insult to the physical personality by distorting the body structure. The reports of cases include seven cases of diseases of the bony framework, such as osteitis deformans and osteogenesis imperfecta, four cases of disturbances in general growth resulting in dwarfism and three cases of diseases of the nervous system that have impaired the motor and sensory function of large parts of the body, such as Little's disease, poliomyelitis anterior and subacute combined sclerosis.

It has been shown that the mechanism of the psychoses is due to definite and readily determined features. These are: (1) the discrepancy between the constitutionally embedded and socially determined concept of the body image or postural model and the actual physical personality or body structure determined by the pathologic process; (2) the mysteriousness and obscurity of the disease which is not understood by the patient and not experienced by him in any one but himself; (3) the thwarting of the adult social, industrial and heterosexual adaptations, which are the strivings that are patterned after the normal postural model but cannot be realized by the distorted body; (4) coincidental confusional or toxic symptoms that arise from the toxic features of the disease process or from pressure by distorted skulls on the brain or nerves, especially when deafness is involved.

I have shown that the psychoses are characterized by three features: (1) specific symptoms of imperception of the body defect or discrepancy between the body image and body structure; (2) emotional upsets due to episodic or accumulative thwartings of the libido; (3) paranoid delusions, symptoms related to the feelings of mysteriousness of the pathologic process. Any of these symptoms may be modified or exaggerated by the confusional and toxic features arising secondarily in connection with the pathologic process.

Although in many of these cases a diagnosis of schizophrenia or dementia praecox has been made, it does not seem that these psychoses belong to this category. There is little evidence of meaningless dissociation in thought processes and none in behavior or emotional responses. Neither are there any catatonic features. Dementia has not occurred when it could not be accounted for by coincidental arteriosclerosis or other organic features.

There is a definitely indicated therapeutic approach to these psychoses based on the foregoing analysis of the mechanism leading to the psychoses. The first treatment, of course, should be directed at the somatic pathologic process in an effort to alleviate the body distortion as much as possible and bring the body structure as near as

possible to the normal body image. The patient is entitled to as full an understanding of the pathologic process as science can make available, and probably, when possible, he should become acquainted with the disease through others who are suffering from it. Every element of mystery and obscurity should be removed. He should then be aided to face and accept the disease and to plan some adaptation to life that will give him a maximum of libido satisfaction. This can be done not only by increasing modes of expression and adaptation, but by training and encouraging a more simple life routine. From time to time hospitalization will offer this simpler mode of life in which the patient may have long periods of contentment that are not otherwise possible.

Diseases that distort the body structure, such as diseases of the bony framework, diseases of growth and diseases of the nervous system that impair the motor and sensory function of large masses of the body may lead to psychoses of a more or less specific type. The psychoses result from the discrepancy between the constitutionally and socially acceptable body image and the distorted body structure; this leads to specific symptoms of imperception of the defect or discrepancy, to thwartings of the adult libido with emotional upsets, to feelings of mystery and obscurity of the poorly understood pathologic process resulting in paranoid delusional systems and to coincidental confusional or toxic symptoms that may be related to the individual pathologic processes. The psychoses do not appear to belong to the category of dementia praecox or schizophrenia, and they offer a therapeutic approach.

Clinical Notes

NEURALGIAS OF THE GLOSSOPHARYNGEAL NERVE

With Particular Reference to the Sensory, Gustatory and Secretory Functions of the Nerve

FREDERICK LEET REICHERT, M.D., SAN FRANCISCO

Neuralgia of the ninth or glossopharyngeal nerve was described clearly by Weisenburg [1] in 1910, but it was such a clinical rarity that when Dandy,[2] in 1927, reported two cases successfully and permanently cured by intracranial division of the nerve central to its ganglia, he was able to collect from the literature records of only eighteen other cases. Keith,[3] in 1932, added seventeen reported cases. Since, then Clinton and Ward,[4] Chavany and Welti,[5] Ball [6] and Reichert [7] have reported six more cases, making a total of forty-three cases recorded in the literature.

The published reports of cases have led to a wider dissemination of knowledge concerning the characteristic symptoms of this rather rare tic douloureux. As a consequence recognition of it is becoming more frequent and more certain, so that a correct diagnosis and successful treatment may soon be expected in every case. Nearly all the reported cases were incorrectly diagnosed originally and treated as neurasthenia, hysteria and, particularly, as trigeminal tic douloureux.

The outstanding characteristic of all primary neuralgias of the ninth nerve is a ticlike pain induced by eating, talking, swallowing or by some movement of the pharynx or tongue. The lancinating, paroxysmal pain is unilateral, and nearly always starts in the tonsillar region, lateral pharyngeal wall or base of the tongue, frequently radiating deeply in the ear. Salivation during paroxysms has been recorded in some cases. In an occasional case the pain may start in the ear, being initiated by swallowing or by other movements of the pharynx and tongue, and radiate to the tonsillar region. Occasionally movements of the ear have

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1. Weisenburg, T. H.: Cerebello-Pontine Tumor Diagnosed for Six Years as Tic Douloureux: The Symptoms of Irritation of the Ninth and Twelfth Cranial Nerves, J. A. M. A. **54**:1600 (May 14) 1910.

2. Dandy, W. E.: Glossopharyngeal Neuralgia (Tic Douloureux): Its Diagnosis and Treatment, Arch. Surg. **15**:198 (Aug.) 1927.

3. Keith, W. S.: Glossopharyngeal Neuralgia, Brain **55**:357 (Sept.) 1932.

4. Clinton, F. S., and Ward, B. W.: Glossopharyngeal Neuralgia, J. Oklahoma M. A. **25**:194 (May) 1932.

5. Chavany, J. A., and Welti, H.: Glossopharyngeal Neuralgia: Symptomatology and Surgical Therapy, Presse méd. **40**:999 (June 25) 1932.

6. Ball, L. H.: Glossopharyngeal Tic Douloureux, Australian & New Zealand J. Surg. **2**:94 (July) 1932.

7. Reichert, F. L.: (*a*) Glossopharyngeal Neuralgia, West. J. Surg. **39**:347 (May) 1931; (b) Three Cases of Glossopharyngeal Neuralgia Cured by Intracranial Section of the Nerve, S. Clin. North America **13**:193 (Feb.) 1933.

started paroxysms. Trigger points have been located in the tonsillar fossa or at the angle between the tonsillar pillar and the base of the tongue.

Glossopharyngeal tic douloureux is fully as severe and as incapacitating as trigeminal tic douloureux with which it has been confused, and relief by intracranial section of the nerve has been immediate and permanent without leaving any physical handicap, since the unilateral numbness of the nasopharnyx, soft palate, posterior pharyngeal wall and tonsillar region and the loss of taste and of sensation over the posterior third of the tongue are not noticed by the patient except on clinical test.

The history and operative results in three cases of primary tic douloureux of the ninth nerve have been recorded elsewhere.[7b] I shall present them only briefly here.

REPORT OF CASES

CASE 1.—J. M. S., a man, aged 53, who was referred by Dr. S. V. Christierson, in 1926 began to have stabbing pain referred to the gums on the right side of the lower jaw just medial to the last molar tooth, which was initiated by laughing, spitting, talking or eating. Saliva drooled from his mouth when he was seen in June 1930; he was unable to talk, but indicated that he had pain in the right side of the throat and jaw. He had not eaten for three days. Previous injections of alcohol and one by me into the mandibular branch of the trigeminus nerve gave no relief, so that the diagnosis of glossopharyngeal tic douloureux was forced on me; this was confirmed by cocainization (10 per cent) of the tonsillar region and base of the tongue, which caused immediate cessation of the paroxysms. A trigger zone was found to include the right tonsillar fossa and base of the tongue. On July 3, 1930, under local anesthesia, the ninth nerve on the right side was sectioned intracranially. The nerve was adjacent to and touched the upper fibers of the tenth nerve at the pons. Touching the ninth nerve alone produced paroxysms and made the ear feel as if it were being irrigated with cold water. In freeing the nerve from the tenth nerve the pulse rate suddenly slowed to 40. This bradycardia persisted for the subsequent six days, and difficulty in swallowing large boluses of food and relaxation of the right palatal arch persisted for nearly a year. When the patient was seen three and a half years after operation, there still remained the same loss of sensation over the right side of the soft palate, pharynx, tonsillar region and posterior third of the tongue, as well as a small area of diminished sensation on the concha, which had been present after operation. Taste was lost over the right posterior portion of the tongue. Simultaneous measurements of the rate of salivary secretion made two and a half years after section of the ninth nerve showed: 0.6 Gm. of secretion from the right and 0.9 Gm. from the left parotid gland, and 0.2 Gm. from the right and 0.25 Gm. from the left submaxillary gland.[8]

CASE 2.—Mrs. M. J., aged 34, complained of recurring attacks of lancinating pain in the left tonsillar region for one and a half years. The pain radiated to the base of the tongue and into the left ear. During the paroxysms the external auditory meatus was sensitive to touch in the lower anterior portion. The attacks were induced by swallowing, eating, drinking cold liquids and talking. The left posterior part of the pharynx and the posterior third of the tongue were hypersensitive, and typical paroxysms could be induced by touching the left tonsillar region. Cocainization of the tonsillar region and base of the tongue

8. Reichert, F. L., and Poth, E. J.: Pathways for the Secretory Fibers of the Salivary Glands in Man, Proc. Exper. Biol. & Med. **30**:973 (April) 1933.

gave complete relief during an attack. On Sept. 4, 1931, under avertin and local anesthesia, the left ninth nerve was divided intracranially. The patient has been free from attacks for the twenty-seven months since operation. Examination after section of the nerve showed sensory loss over the left side of the soft palate, the left posterior pharyngeal wall from the eustachian tube to just below the epiglottis, and the posterior third of the tongue. There was no loss of sensation in the external auditory canal, and the eustachian tube seemed sensitive. The position of the left side of the soft palate was possibly a little flattened on sounding vowels. As the patient lived at a distance, subsequent studies of the salivary secretions were not feasible.

CASE 3.—Mrs. E. H., aged 35, complained of severe lancinating pain in the left side of the throat at intervals for three years. Since the first attack of knifelike pains in the left tonsillar region in February 1929, the tonsils, teeth, sinuses, sphenopalatine ganglion, and "a psychoneurosis" had been treated without affording freedom from the attacks. The diagnosis of glossopharyngeal neuralgia was made from the history and was confirmed when she returned during an attack by cocainization of the trigger zone in the upper part of the left tonsillar fossa. During paroxysms salivation was marked, and she experienced a burning sensation in the throat and neck. No pain radiated to the ear, but pressure on the tragus would often shift the pain from the throat to the ear. The ninth nerve on the left was sectioned intracranially under local anesthesia and avertin on Nov. 23, 1931. The patient has been free from pain during twenty-five months since operation. There has been no change in the sensory loss over the left part of the soft palate, the tonsillar region, the left side of the pharynx from the level of the eustachian tube to just below the level of the epiglottis, and the posterior third of the tongue. There was no change in the position of the palatal arch. Taste was absent over the left posterior third of the tongue. Measurement of salivary secretions [8] obtained sixteen months after section of the nerve intracranially showed: parotid secretion, 0.25 Gm. on the left side as compared with 0.70 Gm. on the normal side; secretion from the submaxillary salivary glands, 0.10 Gm. on the left side as compared with 0.15 Gm. on the normal side.

In addition to these cases of typical neuralgia of the glossopharyngeal nerve, I present one of neuralgia of this nerve limited to the tympanic branch.[9]

CASE 4.—Miss R. S., a telephone operator, aged 31, who was referred to the Stanford Clinic by Dr. D. Carson, in 1921 experienced pain in the left concha which caused her to dispense with the left ear phone for a time. In the spring, 1932, before the onset of her present complaint, she noticed a sensation of drawing and discomfort in the upper half of the left side of the face. Early in August this sensation involved the cheek, forehead and occipital region. A coryza at this time was followed in two days by sharp, stabbing pains deep in the external auditory canal, causing her to shriek and seize hold of her ear. Except for an interval of twelve days, she had recurring daily paroxysms of pain coming without apparent cause, striking deep in the ear and not radiating to any other region. At times there was itching in the external canal with a dull aching of the ear as well as a burning sensation in the left side of the face after an attack. There

9. Reichert, F. L.: Tympanic Plexus (Jacobson's) Neuralgia (True Tic Douloureux of the Ear or So-Called Geniculate Ganglion Neuralgia): Cure Effected by Intracranial Section of the Glossopharyngeal Nerve, J. A. M. A. **100:** 744 (June 3) 1933.

was no salivation during an attack, and swallowing, eating or talking did not induce a paroxysm.

Because of injection and swelling of the posterior superior wall of the external auditory meatus, which was sensitive, efforts were made to eradicate any foci of infection. The sinuses were treated, teeth were extracted, and procaine hydrochloride was injected into the sphenopalatine ganglion and into the sympathetic chain at the seventh cervical and first and second thoracic levels, without influencing the otalgia.

During the first week in December 1932, the paroxysms came every one to three minutes. Sedatives helped little, and the patient begged for relief. As it seemed a true tic douloureux of the geniculate ganglion, operation was advised, and permission was obtained to section the eighth and seventh nerves, if necessary, when sectioning the pars intermedia of the seventh.

The unilateral cerebellar approach was made under local anesthesia on Dec. 10. The left eighth, ninth and tenth nerves were easily identified, and lay separate from one another. When the eighth and seventh nerves were gently touched and moved, the patient stated that she felt pain in the auditory canal, but it was not the tic pain. The ninth nerve was touched, causing her to shriek and exclaim that she had the tic pain. Four times the seventh and eighth nerves were gently moved, and each time she had a pain in the auditory canal; four times the ninth nerve was gently moved, and each time she shrieked because of a stabbing paroxysmal pain in the ear identical with that which had troubled her for four months. The glossopharyngeal nerve was then cut, and the patient fell asleep on the operating table.

Over thirteen months have elapsed since operation and the patient has never felt a suggestion of the tic pain, nor has the pain in the face and occipital region returned.

Following operation there was no anesthesia of the ear or its canal. She located the pain produced by touching the seventh and eighth nerves in the cartilaginous part of the anterior wall of the external auditory meatus, and the tic pain produced when the ninth nerve was touched was referred to the bony part of the anterior wall of the external auditory canal. Sensation, as tested by Dr. E. C. Sewall, was lost over the left side of the soft palate, the pharyngeal wall from 2 cm. within the eustachian tube to the tip of the epiglottis, and the posterior third of the tongue, where taste was also absent.

Ten days after operation the parotid gland [9] on the left side secreted 0.25 Gm., and that on the normal side, 2.2 Gm. Ten weeks after operation the parotid gland on the left side secreted 2.7 Gm., and that on the normal side, 4.5 Gm.; the submaxillary gland on the left side secreted 0.1 Gm., and that on the normal side, 0.9 Gm. Thirteen weeks after operation the parotid gland on the left side secreted 1.05 Gm. as compared with 1.2 Gm. from that on the normal side, and the submaxillary gland on the left side gave 0.1 Gm. as compared with 0.55 Gm. from that on the normal side.

The condition was diagnosed preoperatively as neuralgia of the geniculate ganglion, but at operation, performed under local anesthesia, the ticlike pain was consistently reproduced only when the ninth nerve was touched. The patient had, I believe, a neuralgia or tic douloureux of the tympanic branch (Jacobson's nerve) of the glossopharyngeus; since the symptoms were nearly identical with those in the case which Clark and Taylor [10] reported, the condition in that case undoubt-

10. Clark, L. P., and Taylor, A. S.: True Tic Douloureux of the Sensory Filaments of the Facial Nerve: Cure Effected by Physiologic Extirpation of Geniculate Ganglion, J. A. M. A. **53**:2144 (Dec. 25) 1909.

edly was likewise a neuralgia of Jacobson's nerve instead of a "true tic douloureux of the sensory filaments of the facial nerve." In their patient, in 1909, the eighth and seventh nerves were sectioned intracranially, with relief from a paroxysmal, intermittent, stabbing pain just in front of the ear and in the depths of the ear on the anterior wall of the external auditory canal. At times the patient had a moderate amount of neuralgic pain in all three zones of the trigeminal nerve and in the occipital region. All examinations gave negative results, and several neurologists were forced to conclude that the lesion was a true tic douloureux of the geniculate system of the facial nerve, being guided to this diagnosis by the observations of J. Ramsay Hunt [11] on the zoster zone of the geniculate ganglion. Twelve days after operation, for two hours the patient suffered severe pain in the ear which closely resembled the pain before operation, but subsequently for the six years that she was observed she had no recurrence of the old pain. Mills [12] and Kidd [13] believed that the cure in this case did not prove Hunt's contention of cutaneous sensory fibers in the seventh nerve, and Mills went so far as to suggest, apparently correctly, that "the effects of decompression in such cases should not be overlooked." Although an occasional case of true tic douloureux of the ear has been reported (Harris [14]) operation has not been performed in any case since the famous one reported by Clark and Taylor [10] in 1909. Dandy's [15] patient, although not having a tic douloureux, had a tumor in the external auditory canal causing severe otalgia, which was relieved completely by intracranial section of the ninth nerve. The pain associated with herpes oticus is of a different character, and Hunt's explanation of an inflammation of the geniculate ganglion as the cause of this nonparoxysmal otalgia is convincing.

COMMENT

The ordinary neuralgia or tic douloureux of the glossopharyngeal nerve is characterized by paroxysmal attacks of lancinating pain, usually starting in the tonsillar region or base of the tongue and frequently radiating to the ear, and induced by eating, talking, swallowing or by other movements of the pharynx and tongue. This neuralgia apparently involves the entire distribution of the ninth nerve, causing pain in the throat and ear and producing salivation.

Tic douloureux of Jacobson's nerve is a rare neuralgia of the tympanic branch of the glossopharyngeal nerve, and has been regarded in the literature as a tic of the sensory filaments of the seventh nerve; it is more commonly termed neuralgia of the geniculate ganglion. It is characterized by paroxysms of stabbing pain in the external auditory meatus, often associated with other pains in the face and postauricular region, not induced by talking, eating or swallowing and not associated with salivation.

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11. Hunt, J. Ramsay: Herpetic Inflammation of the Geniculate Ganglion, J. Nerv. & Ment. Dis. **34**:73, 1907.

12. Mills, C. K.: The Sensory Functions Attributed to the Seventh Nerve, J. Nerv. & Ment. Dis. **37**:273 and 355, 1910.

13. Kidd, L. J.: The Alleged Sensory Cutaneous Zone of the Facial Nerve in Man, Rev. Neurol. & Psychiat. **12**:393, 1914.

14. Harris, W.: Neuritis and Neuralgia, New York, Oxford University Press, 1926.

15. Lewis, D., and Dandy, W. E.: The Course of the Nerve Fibers Transmitting Sensations of Taste, Arch. Surg. **21**:249 (Aug.) 1930 (case 3, p. 268).

When operation was performed under local anesthesia for any of these neuralgias of the ninth nerve, the characteristic tic pain was reproduced when the nerve was touched. Fay [16] recently described pain referred to the tonsillar region, the base of the tongue and areas deep in the ear when the hypoglossal nerve or the vagal nerve in the neck had been stimulated by a faradic current, and believed that the hypoglossal nerve carries pain fibers which it receives extracranially directly from the vagus. Whether or not this vagal pain induced by Fay was similar to the paroxsymal type of pain that occurs in glossopharyngeal neuralgia is difficult to state, but it indicates that further observations are necessary to clarify the complicated anatomic and physiologic descriptions of the cranial nerves in the textbooks.

The isolated intracranial division of the glossopharyngeal nerve has permitted a clear differentiation of its functions and has definitely reversed the teachings of Vernet [17] as to the nerve supply of the pharynx.

The observations in ten or more cases of intracranial section of the ninth nerve, in which no injury was inflicted on adjacent nerves, particularly the tenth, are uniform as to the existence of a permanent unilateral anesthesia of the soft palate, the pharyngeal wall from the eustachian tube to the epiglottis, and the posterior third of the tongue. Taste is lost over the posterior third of the tongue; no muscular paralysis can be detected definitely, although the stylopharyngeus muscle is innervated by this nerve.

Poth [8] and I showed recently in three cases of intracranial section of the ninth nerve that there is an immediate and marked diminution in parotid, sublingual and submaxillary salivary secretions followed by a gradual return to almost normal secretion by the third postoperative month. We also observed that when the chorda tympani has been sectioned just distal to its exit from the facial canal, salivary secretion is markedly and permanently diminished. We concluded that the secretory fibers of these glands accompany both the seventh and the ninth nerves. Furthermore, we [18] expressed the belief that the observations of Hesse that parotid secretion is diminished after section of the ninth nerve high in the neck and extracranially, and the results of our examination in a case of peripheral facial palsy with normal taste on the anterior portion of the tongue but with marked diminution of the secretions from the salivary glands, permit only one pathway for the secretory fibers from the ninth nerve to course and unite with those of the seventh nerve in the chorda tympani. This pathway is through an anastomosing branch arising from the trunk of the glossopharyngeus below the petrous ganglion, which joins the facial nerve after the exit of the nerve from the stylomastoid foramen (figure).

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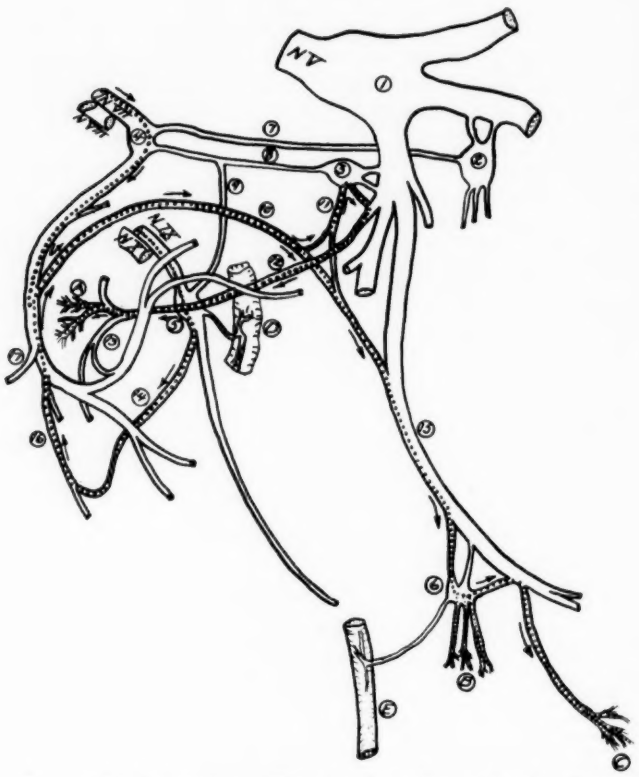
16. Fay, Temple: Observations and Results from Intracranial Section of the Glossopharyngeus and Vagus Nerves in Man, J. Neurol. & Psychopath. **8**:110 (Oct.) 1927; Atypical Facial Neuralgia, a Syndrome of Vascular Pain, Ann. Otol., Rhin. & Laryng. **41**:1030 (Dec.) 1932.

17. Vernet, M.: Syndrome du trou déchiré postérieur (paralysie des nerfs glossopharyngien, pneumogastrique, spinal), Rev. neurol. **34**:117, 1918.

18. Reichert, F. L., and Poth, E. J.: Recent Knowledge Regarding the Physiology of the Glossopharyngeal Nerve in Man with an Analysis of Its Sensory, Motor, Gustatory and Secretory Functions, Bull. Johns Hopkins Hosp. **53**: 131 (Sept.) 1933.



New pathways for the secretory fibers to the salivary glands. Secretory fibers from the ninth nerve pass by an anastomosing branch to the auricular branch of the seventh nerve, then cephalad in the facial nerve to enter the chorda tympani together with the secretory fibers from the seventh nerve. *1* means gasserian ganglion; *2*, Meckel's ganglion; *3*, otic ganglion; *4*, geniculate ganglion; *5*, petrous ganglion; *6*, submaxillary ganglion; *7*, great superficial petrosal nerve; *8*, lesser superficial petrosal nerve; *9*, tympanic nerve; *10*, chorda tympani; *11*, anastomosis between otic ganglion and chorda tympani; *12*, auriculotemporal nerve; *13*, communicating branch of auriculotemporal and facial nerves; *14*, anastomosing branch of glossopharyngeal nerve with digastric branch of facial nerves; *15*, lingual nerve; *16*, digastric branch of facial nerve; *17*, posterior auricular nerve; *A*, parotid gland; *B*, submaxillary gland; *C*, sublingual gland; *D*, internal carotid artery with sympathetic fibers to petrous ganglion; *E*, facial artery with sympathetic fibers to submaxillary ganglion. (From Reichert and Poth: Bull. Johns Hopkins Hosp. **53**:131, 1933).

SUMMARY

Partial and complete involvement of the branches of the glossopharyngeal nerve have led to the differentiation of at least two types of neuralgia of this nerve.

The common or complete tic douloureux of the glossopharyngeus is characterized by paroxysms of lancinating pain starting in the tonsillar region or base of the tongue and frequently radiating deeply in the ear, accompanied by salivation and induced by swallowing, chewing, talking or by other movements of the pharynx and tongue.

The partial (Jacobson's nerve) tic douloureux of the glossopharyngeus is characterized by paroxysms of lancinating pain in and about the external auditory meatus, and is not induced by any movements of the pharynx or tongue. This neuralgia has heretofore been considered as neuralgia of the geniculate ganglion or tic douloureux of the sensory filaments of the seventh nerve. Both the complete and the partial (Jacobson's nerve) tic douloureux of the glossopharyngeus are curable by intracranial division of this nerve.

After intracranial section of the ninth nerve a uniform unilateral loss of sensation over the soft palate, the pharyngeal wall from the eustachian tube to the epiglottis, and the posterior third of the tongue has been found constantly. Taste becomes absent over the posterior third of the tongue.

The salivary secretions from the parotid, sublingual and submaxillary glands show an immediate and marked diminution in secretion which, however, approaches that of the normal side by the third month after intracranial section of the glossopharyngeus.

Clinical Notes

SEVERE ACUTE MENINGO-ENCEPHALOPATHY OF LYMPHOGRANULOMATOUS ORIGIN OCCURRING IN THE COURSE OF HODGKIN'S DISEASE

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The history of the patient recorded here is of unusual interest, since it affords an example of the rare localization in the meninges and the brain of a pathologic process that begins ordinarily as a regional involvement of lymph glands, and though it often becomes more generalized, affecting group after group of lymph glands, as well as the spleen and other organs, seldom involves the neural structure. During recent years, however, more cases have been coming to light in which, in the course of the disease, epidural masses compress the spinal cord and cause paraplegia, and careful clinical studies and investigations at autopsy have revealed occasional involvement of the meninges and of the roots of the cerebral and spinal nerves. In 1933, a Moscow physician, Serebrjanik,[1] described a case that he designated as meningo-encephalitis with polyradiculitis, in which he believed the neural lesions were due to this same pathologic process that predominantly involves the lymph glands and spleen. The case that he reported appears to be the only one recorded in the literature that resembles in localization and in severity the case described here, and the latter presents several features that appear to be unique.

REPORT OF A CASE

History.—Theresa B., aged 14, was admitted to the Osler Clinic of the Johns Hopkins Hospital (service of Prof. Warfield T. Longcope) on Jan. 15, 1934, in a comatose state and with a history of vomiting for the two preceding days.

The parents and one brother were living and well. There was no history of tuberculosis or of any disease resembling that from which the patient suffered in any member of the family. As a child the patient had been frail. In her third year she was brought to the Harriet Lane Home with the history of recurring boils from the age of 3 months. Aside from the furunculosis, she was normal on physical examination; she was treated with staphylococcic vaccine. In later years she was treated for various cutaneous eruptions, including scabies, eczema and impetigo, and once for a febrile attack that was regarded as pyelitis, which cleared up quickly under treatment with sodium bicarbonate. In 1923, she received mild roentgen irradiation for enlarged tonsils and adenoid tissue in the pharynx. From 1924 it was noticed that the glands at the angles of the jaw were enlarged, and later the lymph glands in the left posterior triangle of the neck became enlarged. Though tuberculous lymphadenitis and Hodgkin's disease were thought of, tuberculin tests gave negative results; it was believed that the

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Presented at the Clinic to the Senior Students of the Johns Hopkins Medical School, Feb. 1, 1934.

1. Serebrjanik, B.: Lymphogranulomatöse Meningo-Encephalitis und Polyradiculitis, Deutsche Ztschr. f. Nervenh. **129**:103, 1933.

enlargement of the lymph glands was secondary to infected tonsils and adenoids, and their removal was advised. Owing to phlyctenular conjunctivitis, removal of the tonsils was postponed until October 1929; the operation had no effect on the enlargement of the lymph glands.

During the period from 1930 to 1933, the patient was admitted on five different occasions to the University of Maryland Hospital (service of Professor Pincoffs) where at first the enlarged glands were believed to be due to tuberculous adenitis. She was treated with tuberculin and with ultraviolet rays and was also given three roentgen treatments; after the latter the glands were said to have been greatly reduced in size. There was some secondary anemia (red cells, 3,750,000; white cells, 14,500; hemoglobin, 70 per cent, and polymorphonuclear leukocytes, 76 per cent).

Under tuberculin treatment and repeated roentgen treatment, the glands remained small until November 1931, when there was a recurrence of the enlargement, and bilateral ulceration of the corneas appeared. A biopsy was decided on. The glands were found to be adherent; on microscopic examination no evidence of tuberculosis of the glands was observed, though there was marked hyperplasia of lymphoid cells and of the reticulo-endothelial elements. There were many eosinophil cells in the glands, and occasional cells of the Dorothy Reed type were seen. Dr. Hugh Spencer believed that the histologic aspects strongly suggested Hodgkin's disease. The only glands palpable at that time were in the neck, but the tip of the spleen was palpable on deep inspiration. The temperature varied between 99 and 101 F., and there was moderate polymorphonuclear leukocytosis, with as high as 86 per cent polymorphonuclears. It was concluded that the enlargement of the glands was due to Hodgkin's disease, but it was thought that the conjunctivitis with corneal ulceration and the slightly increased shadows of the roots of the lungs on roentgen examination might be due to tuberculosis. Treatment by roentgen rays in larger doses was continued.

In the autumn of 1932, the patient was admitted to the University of Maryland Hospital for the fifth time because of loss of weight, high fever, severe headaches and cough. The glands on both sides of the neck were enlarged, but the lungs were reported as clear. The spleen was palpable 3 fingerbreadths below the left costal margin, and a mass of intra-abdominal glands was palpable in the left lower quadrant of the abdomen. The leukocyte count was then 18,250, with 80 per cent polymorphonuclears, and the number of blood platelets was found to be increased. The temperature rose as high as 104 F. on three occasions during the patient's stay in the hospital. Roentgen rays were applied to both sides of the neck and to the left lower quadrant of the abdomen. The patient left the hospital against advice. She became worried, began to vomit and on Jan. 15, 1934, could not be roused.

Summary of Physical Examination in the Johns Hopkins Hospital.—The temperature was 97.8 F.; the pulse rate 120 and the respiratory rate 10. The blood pressure was 140 systolic and 80 diastolic. There were marked emaciation, drowsiness and no response to questions except an occasional grunt. There was a slight, dry cough. Brown pigmentation of the skin, which was most marked at sites of previous roentgen treatment, was present. Recurring nystagmus was observed, with conjugate deviation to the right. There were: bilateral corneal scarring, pyorrhea and dental caries, slight but discrete enlargement of the glands at the angle of the jaw, larger glands in the postcervical region, slight enlargement of the glands in the left axilla and slight stiffness but no retraction of the neck. The lungs were normal on auscultation and percussion. The heart was normal except for a systolic blow and tachycardia. The spleen was enlarged to 2 finger-

breadths below the left costal margin. On palpation of the left lower quadrant of the abdomen there was some evidence of thickening, possibly due to scarring from irradiation or to masses of lymph glands. There was absence of the abdominal reflex on the right. A positive Babinski sign was present on the right. There was a marked positive Kernig sign on both sides.

Laboratory Tests.—Blood: Repeated examinations showed leukocytosis varying between 15,720 and 20,600, with a great relative increase of the polymorphonuclear neutrophils (on one occasion 92 per cent), with relative lymphopenia and without eosinophilia. The sugar content was 90 mg., and the nonprotein nitrogen was 100 mg. per hundred cubic centimeters, probably owing to concentration because of the vomiting.

Urine: Albumin, a few hyaline casts and a trace of acetone were present, the last probably being due to the preceding vomiting. A culture was sterile. The output of phthalein was 40 per cent within two hours.

Cerebrospinal Fluid: Repeated examinations showed increased globulin, an increase in the number of cells (on one occasion to 120 per cubic millimeter), with 92 per cent lymphocytes. No tubercle bacilli were found.

Course.—On the fourth day in the hospital the temperature rose to 105 F. and then fell gradually to normal on the seventh day. It remained normal for two days and then rose gradually to 102 F. on the ninth day. The leukocyte count fell from 35,000 on admission to 9,000 on the seventh day, and rose to 20,000 two days later, the polymorphonuclear neutrophils remaining over 90 per cent.

The patient was fed liquids through a Chetwood syringe and was given fluid intravenously as well as by rectum.

The nystagmoid movements, chiefly to the right, continued, as well as the positive Babinski sign on the right, with a poorly sustained right ankle clonus. The abdominal reflex on the right returned, but was feebler than on the left. The Kernig sign was strongly positive on both sides, and there continued to be slight stiffness of the neck. The patient became more restless and irritable, crying and moaning frequently. She babbled incoherently at first, but words were enunciated fairly clearly later; she showed marked perseveration of both speech and movements, repeating the same phrase.

She was examined by the hospital neurologist, Dr. F. R. Ford, on the fourth day in the hospital, when her temperature was 105 F. He found evidence of sensory aphasia, perseveration, apraxia, unilateral involvement of the pyramidal system and possibly bilateral right homonymous hemianopia, but could not be sure of the latter because of the difficulty of testing. He was of the opinion that there must be a focal lesion in the left hemisphere and spoke of the possibility of a tuberculoma or of localized meningitis, though after hearing the report of the biopsy at the University of Maryland Hospital he considered the possibility of a localized granulomatous lesion in or near the left cerebral hemisphere. Examination of the eyes by Dr. Coston revealed pallor of the disks but no choking.

A few days later, the patient became rational, and the psyche was much clearer. The symptoms of sensory aphasia, of hemianopia and of involvement of the pyramidal system disappeared. Urinary incontinence, which had been present, gave way to normal retention. The Kernig sign continued to be positive bilaterally. Vomiting stopped at the end of a few days, and the patient was able to eat better. Tuberculin tests, made by Dr. Bergmeyer, gave negative results.

Diagnosis.—A section of the gland made at the University of Maryland Hospital was obtained, and the pathologist at the Johns Hopkins Hospital corroborated Dr. Spencer's opinion that the section was typical of Hodgkin's disease. The

resident staff (Dr. J. Bordley and Dr. W. H. Barker) gave up the idea of tuberculous meningitis and considered the whole picture to be due to Hodgkin's disease, with lymphogranulomatous invasion of the meninges and of the left cerebral hemisphere, possibly near Wernicke's area and the motor area. Two members of the visiting staff spoke of the possibility of serous meningitis independent of Hodgkin's disease, and some thought that the patient must have had tuberculosis, because of the earlier phlyctenular conjunctivitis and corneal ulceration, even though the results with tuberculin tests had remained negative throughout. This raises the interesting question as to whether phlyctenular conjunctivitis may not sometimes occur in the absence of tuberculous infection.

As macerated lymphogranulomatous glands injected into the brain of the rabbit produce meningo-encephalitis (Gordon biologic test), Dr. Longcope suggested that possibly the injection of some of the cerebrospinal fluid from this patient into a rabbit's brain might also produce this disease, if the virus was present in the fluid. The injection was made, but at the end of ten days no symptoms of meningo-encephalitis had developed in the rabbit.

COMMENT

Since the studies of Sternberg (1898) and of Dorothy Reed [2] (1902) clinicians and pathologists have been in agreement that the histologic changes in lymphogranulomatosis (Hodgkin's disease) are specific and that when they are demonstrable in a lymph gland or in any other tissue of the body the diagnosis of the malady is certain. In the Johns Hopkins Hospital, accordingly, biopsy of an accessible enlarged lymph gland is always resorted to for diagnostic purposes when Hodgkin's disease is suspected. In cases coming to autopsy the diagnosis can often be made from examination of the cut surface of the spleen alone, which gives the appearance of the broken surface of porphyry or of bologna sausage, a picture which is never seen in any other disease.

The rarity of invasion of the central and peripheral nervous system in Hodgkin's disease by the lymphogranulomatous tissue has been commented on by most pathologists and is in accord with our clinical experience that nervous symptoms are relatively infrequent during the course of the disorder. But in some cases of lymphogranulomatosis the nervous system does not remain immune from invasion. Murchison pointed this out in 1922, and Osler, Welch, Longcope,[3] Pancoast and others have reported invasions of the nervous system in Hodgkin's disease. Ginsburg,[4] of New York, in 1927, called attention not only to cases in which the predominant localization was in the nervous system, but also to the fact that in more than 27 per cent of all the cases carefully examined at autopsy at the Montefiore Hospital the nervous system was invaded.

Though direct primary involvement of the nervous system may occur, the neurologic lesions more often follow previous localization of the malady in adjacent organs (glands or bones). Perhaps the most frequent neural involvement is that due to pressure on peripheral nerves and plexuses (causing pains and paresthesias).

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2. Reed, D. M.: On the Pathological Changes in Hodgkin's Disease, with Especial Reference to Its Relation to Tuberculosis, Johns Hopkins Hosp. Rep. **10**:133, 1902.

3. Longcope, W. T.: On the Pathological Histology of Hodgkin's Disease, Bull. Ayer Clin. Lab., 1903, no. 1, p. 4.

4. Ginsburg, S.: Hodgkin's Disease with Predominant Localization in the Nervous System; Early Diagnosis and Radiotherapy, Arch. Int. Med. **39**:571 (April) 1927.

In many cases, however, there is extension of the lymphogranulomatous process from the bones of the spine or from glands near the spine through the intervertebral foramina to the epidural space, with compression of the spinal cord. As a rule, the pathologic tissue does not extend through the dura, but in some cases it does, and it may apparently even originate sometimes in the subdural space or in the leptomeninges.

Primary lymphogranuloma of the central nervous system appears to be of great rarity, but a few cases are on record in which a primary change seems to have occurred in the brain tissue itself or in one or more of the cerebral nerves.

Among the commoner neurologic symptoms met with in the course of lymphogranulomatosis are: (1) epileptiform attacks (often jacksonian in type); (2) disturbances in the domain of the oculomotor, abducens, trigeminal, facial, acoustic, vagus or hypoglossal nerve; (3) symptoms due most often to intoxication of the central nervous system, including headache, disturbances of sleep, deliria and sometimes euphoria; (4) root symptoms in the domain of the spinal nerves (pains, sensory disturbances, paralyses, atrophies and herpes zoster), and (5) meningeal symptoms, with tachycardia, rigidity of the neck, a positive Kernig sign and increase of cells, especially lymphocytes, in the cerebrospinal fluid, with positive results of the Pandy test, but without the presence of tubercle bacilli.

The Cause of Hodgkin's Disease.—This disorder, as Ginsburg [4] and others have emphasized, is "a specific malignant general disease with protean manifestations in which any tissue or organ in any region of the body may become clinically primarily and predominantly affected." It always begins in reticulum.

Sternberg, who made such a valuable contribution on the histology of the disease, advanced the hypothesis that Hodgkin's disease is due to a peculiar form of the tubercle bacillus, and emphasized the fact that true tuberculosis and Hodgkin's disease are often associated in the same patient. His view that it was a modified form of tuberculosis was strongly opposed, however, other workers pointing out that many cases of undoubted Hodgkin's disease certainly show no clinical or histologic evidence of true tuberculosis, that no tubercle bacilli are demonstrable in the typical lymphogranulomatous foci and that inoculation of emulsions of the affected glands into guinea-pigs do not produce tuberculosis. In 1909, Much and Fraenkel described certain gram-positive, nonacid-fast rods or granules in smears from the glands and said that they could not distinguish them from the granular form of the tubercle bacillus. Others reported that they had confirmed this observation, and some asserted that the granules in rows were demonstrable also in sections, but Lubarsch strongly opposed the view that these rows of granules represented a special form of the tubercle bacillus.

In this country, Bunting and Yates reported a diphtheroid organism in the affected glands and thought that they could produce lesions in monkeys similar to those of Hodgkin's disease by injection. Their results, however, have not been corroborated.

Greater hope for the solution of the problem of the etiology has followed the investigation of a group of workers at St. Bartholomew's Hospital during the past five years (Rose Research Foundation [4a]). They believe that they have demonstrated the presence of a definite pathogenic agent (apparently a virus) in lymphogranulomatous glands. Under aseptic precautions they made a 10 per cent suspension of the gland tissue in broth and kept it in a refrigerator for a week, after which 0.4 cc. of the suspension (proved to be free from bacteria) was

4a. Horder, Thomas, and others: Rose Research on Lymphadenoma, New York, William Wood & Company, 1933.

injected into the brain of a rabbit and 0.6 cc. into the marginal vein of the ear of the same rabbit. After from two to six days, the rabbits thus inoculated might show symptoms (retraction of the head, pareses and fits) indicative of meningo-encephalopathy. This is much the form of reaction obtained by intracerebral injection of the viruses of dermal vaccines, herpes and psittacosis.

But rabbits given injections of lymphogranulomatous suspension present a peculiar and characteristic clinical picture: There are muscular rigidity, with incoordination and ataxia, and spastic paralysis of the posterior extremities; the animals lose weight progressively, usually dying in about ten days, though some slowly recover, in which event they are found to be immune on a second dose of the suspension. When the intracerebral injection kills the animal, no bacteria are found in the brain, but there is evidence of what has been designated as "lymphocytic meningitis," as well as of perivascular infiltration of the blood vessels of the cortex (cuffs of small mononuclear cells).

The investigators were able to produce this meningo-encephalopathy with material from nineteen of twenty patients with Hodgkin's disease, whereas in forty control cases in which material from patients with chronic adenitis or with hyperplasia, leukemia, sarcoma, carcinoma, or tuberculosis of lymph glands was used, negative results were always obtained. The virus is apparently markedly thermostabile, though heat seems to lessen its pathogenicity somewhat. A solution of phenol (0.5 per cent) acting on the material in the incubator for twenty hours at 37 C. kills tubercle bacilli, colon bacilli and pyogenic cocci, but does not kill the lymphogranulomatous virus or the virus of vaccinia or that of psittacosis. It is not yet certain whether the virus is filtrable.

These researches of the English observers afford strong evidence that the causative agent in Hodgkin's disease is a specific micro-organism belonging to the so-called virus group, that it is capable of producing meningo-encephalopathy in rabbits and also in guinea-pigs, that it belongs to the group of thermostabile viruses (like the heat-resisting viruses of trench fever and hog cholera), that it is not present in conditions other than Hodgkin's disease, though it is constantly present in lymphogranulomatosis, and that it can be a help in the diagnosis of the disease in atypical forms, since injection of the suspected lymph gland in suspension causes meningo-encephalopathy in rabbits (biologic test of Gordon).[4b]

Portal of Entry of the Virus.—The fact that the cervical lymph glands are most often earliest involved in the disease is suggestive of entrance in such cases from the nasopharynx. When the intrathoracic glands are first involved, entrance of the virus through the air passages must be considered as probable. In primary involvement of the uterus or other female genitalia the portal of entry may be the vagina. In the rare cases in which the gastro-intestinal tract is first and predominantly involved, the entrance of the virus through the mucosa of the stomach or intestines seems probable. That the virus may sometimes be transmitted through the blood stream itself seems possible because of the occurrence (though rare) of primary lymphogranulomatosis within the brain substance or within an internal organ (for example, the spleen).

That there is true infection or parasitic invasion in Hodgkin's disease seems certain; the changes that occur in different sites in the body must be due to the local action of the virus reaching those sites, and all evidence is against a neoplastic nature of the disease with metastases.

4b. Ogilvie, R. F., and van Rooyen, C. E.: Case Demonstrating Value of Gordon's Test in Hodgkin's Disease, Lancet **2**:641, 1933.

Treatment of Lymphogranulomatosis.—Though the favorable effect of arsenical preparations in Hodgkin's disease has long been known, there seems to be universal agreement at present that the best results are obtained by systematic exposure to roentgen rays or radium. The rays should be applied over the tissues that are predominantly affected, as the best results seem to be obtained in this way. Recently, an effort has been made to treat the body as a whole by feebler roentgen ray dosage coming from a distance (total teleroentgenotherapy of Marchal and Mallet [5]), but this method does not appear to have any very great advantage over repeated regional applications.

Though marked diminution in the size of the affected lymph glands and of the spleen can, in nearly all cases, be brought about by radiotherapy, the time comes sooner or later when this does not prevent the progress of the disease to a fatal termination. When the nervous system is involved, early irradiation is very important, since after fibrotic changes have occurred it is no longer efficacious.

Since the English observers found that a rabbit that recovers after intracerebral inoculation with the virus of meningo-encephalopathy is immune from a second attack when more of the suspension of the virus is injected, one may hope that experiments in the future will yield either an immune serum, on the one hand, or a vaccine made from an attenuated virus, on the other, that may be efficacious in combating this dire disease.

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5. Marchal, G., and Mallet, L.: Sur le traitement de la maladie de Hodgkin par les irradiations généralisées (téléroentgenthérapie totale), Sang **7**:748, 1933.

EPENDYMOMA OF THE FILUM TERMINALE

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The rarity of reports on tumors of the filum terminale may be due to a certain extent to the neglect with which this structure is usually treated in routine necropsies. Embedded as it is in the roots of the cauda equina, small tumors of this structure, such as the one encountered in the case to be reported, may readily escape notice unless a careful examination of this portion of the spinal cord is made.

The filum terminale is the slender glistening prolongation of the end of the conus medullaris. The boundary between the cord and the filum is usually ill-defined; it may, however, be determined by a dilatation of the central canal of the spinal cord known as the ventriculus terminalis. At about the level of the second sacral vertebra the filum terminale internum (intradural filum terminale) undergoes a transition to the filum terminale externum as the dura mater becomes closely applied to it. The filum terminale externum (extradural filum terminale), sometimes called the coccygeal ligament, is then directed toward the base of the coccyx to which it is attached.

The filum terminale has been known to contain astrocytes, oligodendrocytes, ganglion cells, axis-cylinders and numerous ependymal cells. The case reported here, as well as previous sections obtained from normal material, afforded the opportunity of demonstrating microglia in the filum terminale internum, thus completing the analogy in interstitial cells to the brain and spinal cord. Tumors of the filum terminale may therefore be of histologic types encountered elsewhere in the central nervous system. As the filum terminale externum is approached, cellular elements become less numerous and ependymal cells relatively more prominent. The cells are ultimately replaced by strands of connective tissue comprising the coccygeal ligament.

Tumors arising from the filum terminale externum have not been extensively studied, although it is possible that they form an appreciable proportion of the group of sacral tumors which includes practically all types of growths. Moersch [1] in an analysis of forty-two cases of sacral tumors observed at the Mayo Clinic found ten low-lying ependymal-cell gliomas, four occurring at a higher level of the sacrum. Mallory [2] described an ependymoma on the back of the coccyx in a woman, aged 44, which had been first noticed twenty-five years previously. He believed that the tumor had arisen from embryonic remains of the lower end of the neural canal, the closure of which is irregular in this region. The occurrence of gliomas outside of the usual limits of the central nervous system may be related to developmental anomalies. On the various cerebrospinal nerves, for

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Read before the Chicago Neurological Society, Dec. 21, 1933.

From the Division of Neurology and Neurosurgery, University of Chicago Clinics.

1. Moersch, F.: Sacral Tumors, Report of Forty-Two Cases with Special Reference to Their Neurologic Aspects, M. Clin. North America **10**:715, 1926.

2. Mallory, F.: Three Gliomata of Ependymal Origin: Two in the Fourth Ventricle, One Subcutaneous over the Coccyx, J. M. Research **18**:1, 1902.

example, I have frequently encountered groups of glia cells (astrocytes and oligodendrocytes) as well as nerve cells several millimeters distal to the site of transition from the central to the peripheral nervous systems.

This report is concerned with a tumor of the filum terminale internum. Gowers,[3] in 1876, described a myolipoma in the region of the filum terminale attached to the conus medullaris and apparently springing from the pia mater. This tumor was encountered accidentally in a case of tabes dorsalis. Other tumors arising from the filum terminale internum have since been reported by Lachman,[4] Gaupp,[5] Spiller,[6] Zaufal [7] and Sachs, Rose and Kaplan.[8] Kernohan [9] and Kernohan, Woltman and Adson [10] reported eighteen gliomas of the filum terminale and three hemangioblastomas. They classified eleven of the gliomas as ependymoma, three as astroblastoma, two as spongioblastoma multiforme, one as astrocytoma and one as oligodendroglioma. Seven additional gliomas involving both the conus medullaris and the filum terminale were recorded by them. In addition to the growths in the aforementioned cases, it is probable that some of the extensive tumors occurring in the region of the cauda equina arise from the filum terminale, although their exact origin is often difficult to ascertain. The tumor described by Saxer [11] as very likely of ependymal origin may well belong to this category.

REPORT OF CASE

History.—This report concerns a white man, aged 54, who was seen first at the University of Chicago Clinics on Nov. 9, 1933, suffering from the following complaints: sacral pain radiating down the posterior aspects of both lower extremities, of seven years' duration, aggravated by coughing, sneezing or straining at stool, abdominal distress and constipation of one year's duration and mild urinary hesitancy.

Examination.—The general physical examination gave negative results. On neurologic examination slight hypalgesia, hypesthesia and thermohypesthesia over the second, third, fourth and fifth sacral segments, with some hyperesthesia to all

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3. Gowers, W. R.: Myolipoma of Spinal Cord, Tr. Path. Soc. London **27**:19, 1876.

4. Lachman, B.: Gliom im obersten Teil des Filum terminale mit isolierter Compression der Blasennerven, Arch. f. Psychiat. **13**:50, 1882.

5. Gaupp, J.: Casuistische Beiträge zur pathologischen Anatomie des Rückenmarks und seiner Häute, Beitr. z. path. Anat. u. Physiol. **2**:510, 1888.

6. Spiller, W. G.: Lipoma of the Filum Terminale, J. Nerv. & Ment. Dis. **26**:287, 1899; in discussion of Sachs, Rose and Kaplan.[8]

7. Zaufal, G.: Ueber einen Tumor des Filum terminale, Centralbl. f. allg. Path. u. path. Anat. **9**:385, 1898.

8. Sachs, E.; Rose, D. K., and Kaplan, A.: Tumor of the Filum Terminale with Cystometric Studies, Arch. Neurol. & Psychiat. **24**:1133 (Dec.) 1930.

9. Kernohan, J.: Primary Tumors of the Spinal Cord and Intradural Filum Terminale, in Penfield: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3.

10. Kernohan, J.; Woltman, H., and Adson, A.: Gliomas Arising from the Region of the Cauda Equina, Arch. Neurol. & Psychiat. **29**:287 (Feb.) 1933.

11. Saxer, F.: Ependymepithel, Gliome und epitheliale Geschwülste des Centralnervensystems, Beitr. z. path. Anat. u. Physiol. **32**:276, 1902.

forms of sensation at the eleventh thoracic segment were found. A lumbar puncture revealed normal cerebrospinal fluid under normal dynamics. One cubic centimeter of iodized poppy-seed oil 40 per cent was injected in the fifth lumbar interspace. When a fluoroscopic examination was made with the patient's head down the iodized oil was arrested at the second lumbar vertebra; when the patient was in the upright position it descended to the second sacral vertebra. A presumptive diagnosis of tumor of the cauda equina was made and an operation advised.

Operation.—Laminectomy of the first, second and third lumbar vertebrae by Dr. Bucy disclosed only a small tumor (fig. 1 *A*) of the filum terminale a short distance below the conus medullaris. The growth was removed, although it did not seem large enough to be responsible for the symptoms. A small rubber

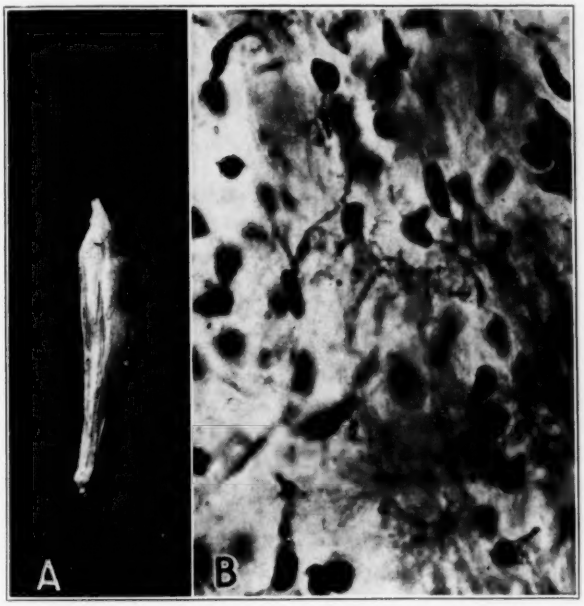

Fig. 1.—*A*, tumor showing the short end of the filum terminale toward the conus medullaris and the longer portion of filum terminale distally. Frayed fibers of the filum terminale pass ventrally, the tumor presenting chiefly dorsally. *B*, filum terminale adjacent to the tumor showing microglia as elongated granular cells with spinous processes. Early transitional forms to compound granular corpuscles, fragmentation of processes with swelling of the cytoplasm and an approach toward the rod cell (cell in the lower left corner) are seen. Impregnation with lithium-silver carbonate stain; × 800.

catheter introduced into the subdural space was passed from 12 to 15 inches (30 to 38 cm.) upward without encountering obstruction. Exposure of the cauda equina to the sacrum revealed no further abnormality.

Course.—The convalescence was uneventful except for difficulty in voiding, necessitating catheterization, with transitory cystitis. Four roentgen radiations of 312 roentgens each were administered over the lower lumbar and sacral parts of the spine on the assumption that there might be some lumbosacral radiculitis,

although at operation the roots of the cauda equina appeared normal. The patient was discharged three weeks after the operation; when seen two weeks following discharge he had no pain, and examination revealed normal sensation for all modalities. He has since remained perfectly well. The case is presented because of the interesting pathologic condition, the small size of the tumor permitting study serially with a loss of but a few sections.

Histology.—Gross Description: At operation the tumor appeared as a small fusiform swelling of the filum terminale (fig. 1 *A*), beginning about 1 cm. below the termination of the conus medullaris. The nerve roots of the cauda equina were not adherent to it. The tumor was removed with about 6 mm. of filum terminale attached to its lower pole. It weighed in all 0.1 gm. after partial fixation in formaldehyde, and measured 13 mm. in length and 4 mm. in diameter. It presented chiefly posteriorly, the white glistening frayed fibers of the filum terminale enveloping mainly its anterior half. It was yellowish, nonlobulated and enclosed in a distinct fine pia-like membrane with occasional thickened fibrous bands of the filum terminale passing over it. There were but few small blood vessels passing over the surface of the tumor, the anterior spinal vessels being the most prominent and not enlarged. Along the anterior aspect of the tumor at its lower junction with the filum terminale lay a soft brownish area, 2 mm. in diameter, representing old hemorrhage. The tumor was soft and fatlike. Longitudinal section after fixation in formaldehyde revealed a dull gray nonvascular tumor speckled with small whitish areas merging with an oval, 3 by 1 mm., yellowish core. No fibrous strands were visible within the tumor. The brownish zone of hemorrhage faded gradually a few millimeters along the anterior portion of the tumor.

Following longitudinal section of the tumor one half was embedded in paraffin and sectioned serially along its long axis. These sections, 5 and 10 microns thick, were stained by Laidlaw's silver-impregnation method for connective tissue and with hematoxylin and eosin, hematoxylin and van Gieson's stain, mucicarmine stain, Mallory's phosphotungstic acid stain (Davidoff's modification), Mallory's ammonium sulphide stain for iron pigment and Weigert's elastic tissue stain. The other half of the tumor was sectioned on the freezing microtome. Many of these sections, cut at 15 microns, were stained with scarlet red for fat and with thionine for Nissl substance; the Gros-Bielschowsky technic was employed for nerve fibers and the Weigert-Pal method for myelin sheaths. Other sections were placed in a solution of formaldehyde and ammonium bromide and after a week or longer of further fixation the usual neuroglial methods were employed. The only satisfactory preparations obtained were on material impregnated with del Rio Hortega's washed lithium-silver carbonate stain (2.5 per cent) exposed at 56 C. for about one-half hour before reduction in a solution of formaldehyde. Microglia, astrocytes, astroblasts, spongioblasts and occasional oligodendrocytes were thus impregnated. Del Rio Hortega's first variant as well as Mallory's phosphotungstic acid-hematoxylin stain was used for blepharoplasts.

Microscopic Description: The tumor was composed of fairly large polygonal cells with central nuclei containing one or more well defined nucleoli, usually centrally placed in a moderate chromatin mesh. The nuclei were frequently vacuolated and tended to form clumps with an apparent fusion of cytoplasm, occasionally suggesting giant cells (fig. 2 *A*). Mitotic figures were not found.

The cells formed a mosaic of sheets, clusters, single or multiple rows and complete or incomplete rings of varying interrelationship (figs. 2 *A* and *B* and 3 *A*). The cell boundaries were not always distinct. Cytoplasm was abundant, and there was often a clear space around the nucleus. Within the cytoplasm were granula-

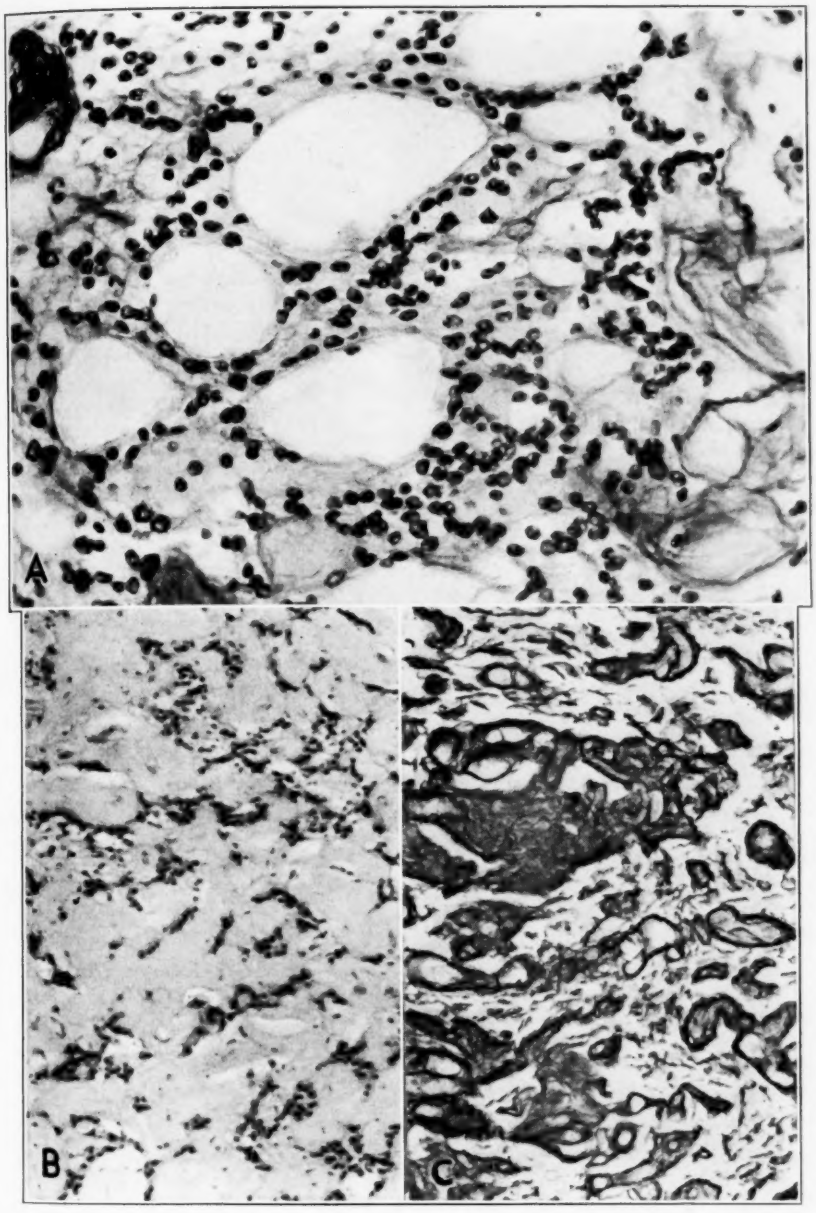

Fig. 2.—*A*, ependymal rings with nuclei showing vacuolation. Few nucleoli are seen in this section; other stains demonstrate nucleoli in practically all the cells as in the adjacent ependymal cells of the filum terminale. A tendency toward clumping of nuclei suggestive of giant cells is seen. Vacuolated cytoplasm of many of the cells represents dissolved out fat stained abundantly with scarlet red. Hematoxylin and van Gieson stain; × 300. *B*, less cellular pattern of the tumor resulting from the proliferation of its stroma and degeneration. Intercellularly as well as intracellularly to a slight extent, mucin is stained with mucicarmine. Hematoxylin and eosin; × 100. *C*, field similar to that of *B* showing abundant reticulin stroma continuous with that of the surrounding pia mater capsule and the blood vessels. Balls of reticulin or blood vessels are surrounded by tumor cells. Laidlaw's connective tissue stain; × 100.

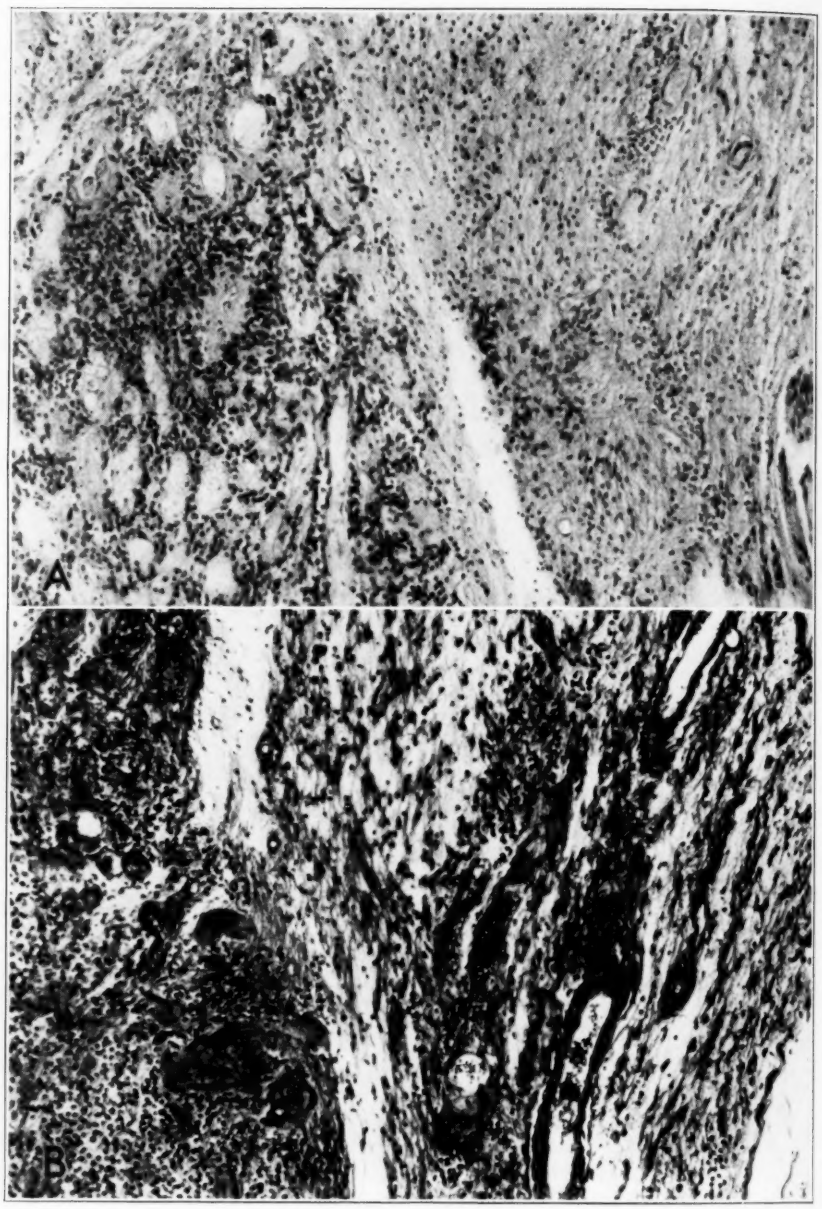

Fig. 3.—*A*, tumor on the left and neuroglial portion of the filum terminale on the right containing a few islands of ependymal cells resembling those of the tumor. The rosette in the tumor is surrounded by a few ependymal rings. Hematoxylin-eosin; × 100. *B*, solid cellular pattern of the tumor is seen on the left with the filum terminale on the right. Gliosis is seen in the adjacent filum terminale in the midportion of which few ependymal cells resembling the type cell of the tumor occur. Higher magnification reveals some differentiation of spongioblasts to astroblasts and astrocytes in the marginal zone of the tumor. Hypertrophy of astrocytes is seen on the right. Impregnation with lithium-silver carbonate; × 100.

tions confined to one or several portions of the cell. They appeared as round bodies varying in size from that of mere dots to that of large cocci, occasionally surrounded by halos.[12] They usually formed groups of two, as many as twenty, however, being found in some cells; in a few instances these markings were located at opposite poles of the cells arranged around blood vessels. The same granulations were seen in the ependymal cells of the adjacent filum terminale. They were also present in spongioblasts and in the more adult neuroglia cells of the tumor. Similar bodies were simultaneously stained in astrocytes of the neighboring filum terminale; in such instances they occurred in the perinuclear cytoplasm and in that of the cell expansions. They were probably gliosomes. They could at times be mistaken for fibrils seen in cross-section. These granulations within the astrocytes could usually be differentiated from the markings of ependymal cells by the fact that they did not exhibit the tendency toward grouping seen in the latter and were not surrounded by halos; also, they seldom formed the larger round or rod-shaped bodies seen in ependymal cells.

The groups of cells were separated by an abundant faintly eosin-staining substance. Specific methods of impregnation identified this intercellular material as reticulin, some of which manifested a tendency toward hyalinization. The reticulin stroma, containing only occasional fibroblasts, was continuous with that of the few thin-walled blood vessels and the connective tissue capsule (fig. 2*C*). There was no evidence of endothelial proliferation. Elastic tissue was present only within the walls of the few larger blood vessels. Some of the intercellular substance had undergone degeneration, staining specifically with mucicarmine. Mucin was present intracellularly as well in the form of droplets, singly or in clusters. This material was present within the tumor cells. Scarlet red stained fat within these cells as well as abundantly in large phagocytes; these could frequently be differentiated from the tumor cells by their smaller nuclei which contained more chromatin. Old blood pigment was present at the margin of the tumor adjacent to the filum terminale. It occurred in phagocytes and apparently lay free in tissue spaces and blood vessels. No areas of massive necrosis were seen. Calcification did not occur.

The rings of tumor cells enclosed clear canals, reticulin balls, clefts containing a little débris or blood vessels whose walls were possibly hyalinized. Such cells were either cuboid or cylindric rather than polygonal as in the regions of mosaic. No definite cilia were seen. Resting on the vessel was a limiting membrane formed by the cuticulae of the tumor cells. These cells were at times suspended between blood vessels with one expansion passing to each; at times they were flattened among adjacent blood vessels or balls of reticulin. The cell bodies or expansions at times formed a continuous barrier applied to these structures of connective tissue. Fibrillae could occasionally be seen within the expansions of tumor cells abutting vessel walls. These, however, lacked the cleancut appearance of the intracellular fibrils of astrocytes. Broad expansions, at times branched, projected peripherally as a sheet of cytoplasm with a frayed edge or tapered off gradually. These cells represented ependymal spongioblasts. Cytoplasmic granulations were frequently seen in peripheral as well as in central processes. Many of the neoplastic cells in the vicinity of blood vessels tended to be elongated, their central processes resting against the vessel wall. The peripheral process was at times

12. Bailey, P.: A Study of Tumors Arising from Ependymal Cells, Arch. Neurol. & Psychiat. **11**:1 (Jan.) 1924; Quelques nouvelles observations de tumeurs épendymaires, Ann. d'anat. path. **2**:481, 1925.

quite long, suggesting the supportive spongioblast. Occasionally the arrangement of cells resembled a rosette with one or more small blood vessels at the center (fig. 3 *A*). These rosettes were "distinguished from those of the retinal gliomas by having a blood vessel in the center instead of a cavity."[13] Within the tumor were fine fibrils resembling the intracellular fibrils of the astrocytes of the filum terminale. Certain of these fibrils belonged to the adult astrocytes of the tumor; similar fibrils, though usually coarser, represented expansions of many of the tumor cells.

The tumor was surrounded by a well defined capsule of connective tissue which was invaded in areas by tumor cells. This tumor sheath was formed of thickened pia mater the reticulin of which was continuous with that of blood vessels and tumor stroma. The anterior surface of the tumor could be identified by the large blood vessels which were normally present in the pia mater anteriorly. Filum terminale was present at both ends of the tumor and along the ventral border. There was no sharp demarcation between the tumor cells and the normal ependymal cells of the filum terminale, which were similar; the cells contained blepharoplasts, nucleoli and an identical chromatin pattern.

At both poles of the tumor the filum terminale, slightly invaded by tumor tissue, presented interesting changes in relation to adjacent neuroglia (fig. 3 *B*). There were areas in which the astrocytes had increased in number, their fibrous processes forming a dense mesh of tangled gliosis. Mitotic figures were not seen in these regions, but evidence of direct division occurred, binucleated astrocytes being present. More distal to the tumor were smaller star-shaped astrocytes, beyond which oligodendrocytes were seen. There were instances of resemblance between some of these small astrocytic forms and oligodendrocytes with their delicately inserted processes. Acute swelling of oligodendrocytes was not encountered. In other regions of the tumor margin phosphotungstic acid stains and silver impregnations revealed polar spongioblasts, astroblasts and astrocytes. These cells were also present within the tumor itself. In these regions the astrocytes were usually round, star-shaped or triangular, with short processes and occasionally demonstrable intracellular fibrils. They lacked the well formed appearance of the astrocytes of the adjacent filum terminale. Some of the astroblasts as well as astrocytes at the border of the neoplasm possessed vascular attachments, at times radiating parallel to the vessel walls or septums of connective tissue. One could not always differentiate these adult forms from adjacent astrocytes of the filum terminale; the fact that intermediate types were present suggested that many of these adult elongated cells were neoplastic. In some areas of neuroglia the presence of microglia assisted in this differentiation. Their occurrence as normal or slightly altered cells indicated adjacent filum terminale rather than tumor tissue.

The tumor was bordered laterally and dorsally simply by a capsule of connective tissue and ventrally in addition by a rather wide zone of filum terminale. In the latter region there was a moderate degree of gliosis; the astrocytes were elongated and hypertrophied, at times binucleated, their fibrous processes being parallel to the axis of the tumor. There was some tendency toward a chronic degenerative change of astrocytes with hypertrophy of cell body and process and gradual loss of expansions associated with dendrophagocytosis. Few monster cells were seen. An opportunity for studying the stages in transformation of microglia to compound granular corpuscles was afforded (fig. 1 *B*). In the vicinity of the adjacent filum

13. Bailey, P.: Histologic Diagnosis of Tumors of the Brain, Arch. Neurol. & Psychiat. **27**:1290 (June) 1932.

terminale normal microglial cells were seen; transitional forms included microglia with swollen elongated bodies, fragmented processes and vacuolated cytoplasm, rod cells and globular scavenger cells with reticulated cytoplasm filled with fat. Many of these compound granular corpuscles and closely allied forms were present within the outer portions of the tumor. Fat cells were seen in the perivascular spaces of the tumor and fat was stained in a few instances within the lumen of the vessel. Scavenger cells could be seen at times in the vicinity of blood vessels adjacent to the tumor, and normal microglial cells also occurred at those sites. This coincidence recalls Penfield's [14] suggestion of a delivery of ingested substance to the blood vessel with liberation of the microglia for renewed phagocytic activity. Ganglion cells surrounded by tumor cells occurred near the border of the neoplasm. These cells were not neoplastic but indigenous to the filum terminale. They exhibited marked chromatolytic change, with dissolution or clumping of Nissl-substance, displacement of the nucleus and even rupture of the cell border. Nissl-substance was not seen in any of the tumor cells. Neurofibrils or axis-cylinders were similarly not stained in the neoplasm. The nerve fibers were confined to the periphery of the tumor as seen in the Gros-Bielschowsky and Weigert-Pal preparations.

COMMENT

Serial section of a small tumor with its surrounding structure gave an opportunity for the study of its origin and composition. The sections of a tumor may vary considerably in architecture and types of cells, at times suggesting divergent diagnoses. Thus, although there were spongioblasts, astroblasts and astrocytes in the tumor studied, the predominant cell was polygonal, containing blepharoplasts and exhibiting a tendency toward ring formation. In cell type, and largely in architecture, the tumor resembled the central portion of the adjacent filum terminale. Accordingly it must be classified as an ependymoma.

The subdivision of ependymomas into various pattern types does not seem advisable, since the pattern may vary from section to section and even within a single section. There were areas of this tumor which were typically composed of ependymal rings similar to the appearance of portions of the normal filum terminale; other areas presented a pattern of solid cells, again recalling the structure often observed in the filum terminale in which the ependymal cells are abundant and crowded together. In addition to the growth of neoplastic cells there was a proliferation of connective tissue stroma derived from the surrounding pial sheath and continuous with that of the blood vessels. When the tumor cells apparently outgrow the limits of their blood supply degeneration occurs which may, as in this case, be fatty or mucinous, and another pattern may thus be produced. Areas of massive tissue necrosis or calcification are not found. Degeneration is rather evidenced by vacuolation of nuclei, by the presence of mucin within the areas of abundant connective tissue or intracellularly, by hyalinization of the connective tissue stroma and by the presence of fat in tumor cells or phagocytes. The phagocytes may be derived from the microglia normally inhibiting the filum terminale or from the other usual sources (blood or vascular adventitia). Stages in the transformation of microglia to compound granular corpuscles could be seen at the border of the tumor. The striking amount of fat within the neoplasm was responsible for its being mistaken grossly for a lipoma.

14. Penfield, W.: Microglia and the Process of Phagocytosis in Gliomas, Am. J. Path. **1**:77, 1925.

A survey of the literature reveals reports of twenty-six tumors of the filum terminale internum. The microscopic description of many of these tumors is inadequate. However, the diagnoses offered permit the following tabulation:

Gliomas		20
Ependymomas	11	
Astroblastomas	3	
Spongioblastoma multiforme	2	
Astrocytoma	1	
Oligodendroglioma	1	
Glioma, unclassified	2	
Case of Lachman		
Case of Saxer (ependymoma?)		
Hemangioblastomas		5
Lipomas		1

The present case of ependymoma brings the total tumors of the filum terminale to twenty-seven, the most common type being the ependymoma (twelve). This frequency is understandable from studies of the normal filum terminale, in which ependymal cells are most abundant.

SYPHILITIC MYELOPATHY

REPORT OF A CASE

THEODORE T. STONE, M.D., AND ARTHUR WEIL, M.D., CHICAGO

Syphilitic myelopathy is a rare acute entity and should not be confused with syphilitic myelitis, which is usually remittent and requires months or years to develop. Syphilitic myelopathy is an acute disease occurring in the secondary stage of syphilis or soon afterward. It is characterized clinically by flaccid paralysis of the lower extremities, loss of sphincteric control and of all forms of sensation below the lesion, spontaneous spasmodic movements, decubitus and ascending pyelocystitis. It is characterized pathologically by diffuse and irregular demyelinization and vascular changes. The prognosis is usually grave. In differential diagnosis, distinction must be made from transverse myelitis of infectious origin and thrombosis of the anterior and posterior spinal arteries.

REPORT OF CASE

History.—M. E., a white man, an engineer, aged 26, single, on Nov. 10, 1932, at about 3 p. m., found himself unable to urinate when he felt the desire and at the same time noticed that walking was an effort and that his legs had become weak. There was no pain or paresthesia. The legs continued to become weaker, and at 11 p. m. he was taken to a hospital because of inability to urinate or walk. There he was catheterized, but the stricture which had been suspected and for which he was admitted was not found. He was unable to walk to the hospital. It was found that if the bladder was allowed to fill, urine would flow spontaneously at times. Incontinence of the bowels occurred at this time. He was seen by one of us (T. T. S.) on November 24. The family history was of no import. The patient had had the usual diseases of childhood. He had had gonorrhea four years previously. He gave no history of a primary syphilitic lesion, but said that he had had a severe sore throat about six months previously which was not accompanied by fever, rash, pains in the bones or joints or alopecia. For many reasons it was evident that this was a syphilitic infection. An inventory by systems failed to reveal any important symptoms.

Examination.—The patient was thin and pale and had a washed-out facial appearance. The temperature was 98.8 F.; the pulse rate was 76, and the respiratory rate 22. There was nonatrophic flaccid paralysis of both lower extremities. The plantar reflexes were absent bilaterally. The achilles jerk was present on both sides; it was slightly diminished, more so on the left than on the right. The knee jerk was present on both sides and was equal and normal. No pathologic reflexes were present. Sensation for pinprick was completely lost anteriorly over both lower extremities, the abdomen and the lower part of the chest to the sixth thoracic segment on the right and the fifth thoracic segment on the left. Posteriorly, sensation for pinprick was lost over both lower extremities, the buttocks and

From the Department of Nervous and Mental Diseases and the Institute of Neurology, Northwestern University Medical School.

the lower part of the back to the eighth thoracic vertebra on the left and the ninth thoracic vertebra on the right. Temperature sense was lost over the same areas both anteriorly and posteriorly, except that this loss extended to a point 2.5 cm. below the level of loss of sensation for pinprick. Sensation for light touch was lost over the same areas, except that the upper boundary reached to the eighth thoracic skin segment on each side. Proprioceptive sensation was lost in both lower extremities and both groins (twelfth thoracic skin segment), except that the patient was able to appreciate bone and joint sense in the left large toe, position sense in the left lower extremity and vibration sense in the left knee. There were many spontaneous spasmodic movements in both lower extremities, chiefly in the hips and thighs. The cremasteric reflex was present bilaterally, but was markedly diminished. The abdominal reflexes were absent bilaterally. The radial and ulnar, supinator, triceps and biceps reflexes were present bilaterally, and were equal and normal. Sensation in all modalities was normal in the upper extremities, neck and

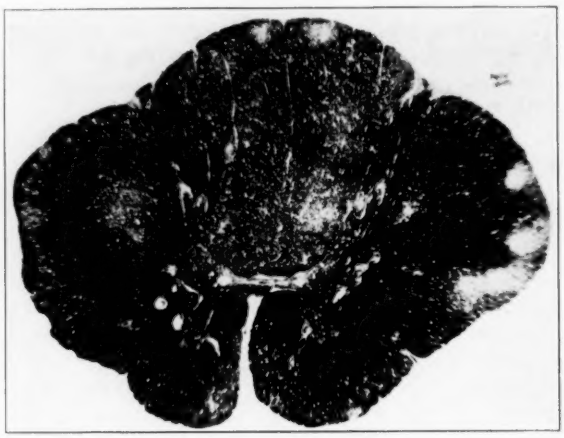

Fig. 1.—Transverse section through the midthoracic region of the spinal cord, showing scattered areas of demyelinization, which are most pronounced at the periphery; Weil stain; Zeiss obj., 1.5 ×; ocul., 6 ×.

head. There was a large decubitus ulcer over the sacrum, one on the right thigh near the trochanter, two small ulcers on the lateral surface of the left foot and one small ulcer on the lateral surface of the left thigh. The cranial nerves were normal, except that the pupils were slightly irregular; they were 3 mm. in diameter and reacted to light with a limited excursion; the reaction consensually and in accommodation was normal. There was no evidence of a psychosis. The Wassermann reaction of the blood was four plus. The spinal fluid was clear; the pressure was 15 cm. of water, and there was no evidence of block. The fluid contained 30 cells, all lymphocytes; globulin and albumin were present. The Lange colloidal gold curve was 2234432100. The Wassermann reaction was four plus.

Diagnosis.—A diagnosis of syphilitic myelitis of the thoracic cord was made.

Course.—The patient was placed on a water bed and received the following treatment from Dec. 17 until Feb. 27, 1933: seventeen inunctions of mercury within nineteen days (these were discontinued because of mercurialism), 1,160 minims of a saturated solution of potassium iodide over a period of twenty-five days and 2.8

Gm. of neoarsphenamine over a period of two months. The decubitus ulcers were treated aseptically.

The patient made no favorable progress despite the therapy. Severe cystitis, pyelocystitis and cachexia developed. The patient died at 9 a. m. on February 27 of pulmonary edema and circulatory failure.

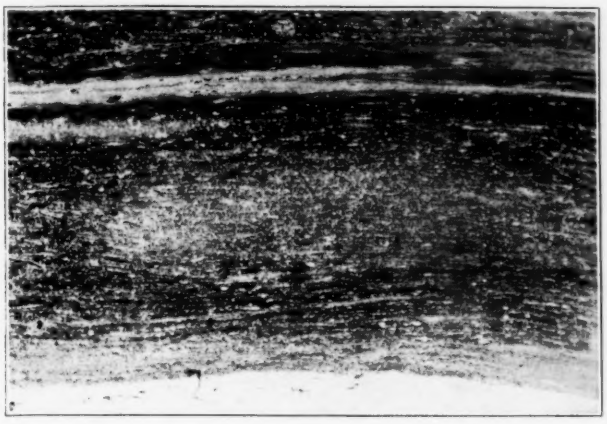

Fig. 2.—Longitudinal section through the lower thoracic segment of the spinal cord, showing rather homogeneous area of demyelinization; Weil stain; Leitz obj., 10 ×; ocul., 6 ×.

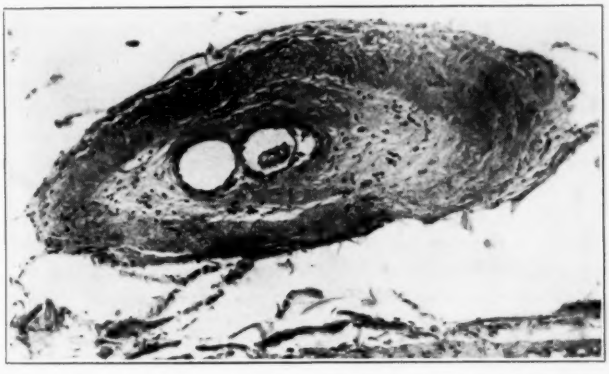

Fig. 3.—Section through the posterior spinal artery of the lower thoracic region, showing proliferation of the intima and degeneration of the media. The elastica interna is barely visible; a double lumen has been formed. Van Gieson stain; Leitz obj,. 10 ×; ocul., 10 ×.

Necropsy.—Only a partial postmortem examination was permitted; several segments of the thoracic cord were removed for study.

Microscopic Observations: Irregularly distributed, patchy areas of demyelinization were seen (fig. 1) in transverse sections of the lower dorsal segments stained for myelin sheaths. They were most marked at the outer periphery and gradually

diminished in size and intensity in the upper lumbar segments. These areas appeared rather homogeneous, or somewhat honeycombed, in longitudinal sections (fig. 2), but nowhere as severely destroyed as in subacute combined degeneration. Within the meshes, fat-laden compound granular corpuscles were seen. The myelin sheaths and axis-cylinders in the environment of these foci were diffusely swollen and partly fragmented. The different glial elements were not severely diseased, and there was no progressive fibrous glial reaction. The demyelinization did not affect the anterior or posterior spinal nerve roots. The normal appearance of the neurons of the anterior and posterior gray matter was distinctly in contrast with that of the severely diseased white matter.

The pia-arachnoid was thickened and mildly infiltrated by scattered small round cells. The walls of the posterior spinal arteries had lost their characteristic histologic structure; the media and the internal elastic membrane had been replaced by a homogeneous mass of connective tissue, which stained intensely red in van Gieson preparations. Occasionally a double lumen or marked narrowing of the lumen by the proliferated tissue was seen (fig. 3). A similar homogeneous degeneration of the vascular walls was seen in the central arteries, around which a mild, slight infiltration by round cells was seen in some sections. In sections stained by the Perdrau method one was impressed by the great number of fibrotic capillaries in the gray matter.

COMMENT

Different pathologic processes seemingly combined to produce the pathologic picture described: mild meningomyelitis; degeneration of the wall of the posterior spinal arteries, with intimal proliferation, and destruction of nerve fibers throughout the white matter of the lower dorsal and upper lumbar segments, with a process of repair in its first stages. Although no spirochetes were demonstrated, the clinical history and the laboratory observations suggested that syphilis was the etiologic factor. The question arises whether there was a direct relationship between the three pathologic conditions or whether they were independently produced by the syphilitic toxin. As the inflammatory reaction occurred only occasionally and was mild, it obviously could not have been responsible for the marked destruction of the white matter. One might think of an anoxemic process (softening) following the narrowing of the lumen of the posterior spinal arteries. A similar process in cerebral arteries in syphilitic disease was described by Alzheimer,[1] as "hyalin-sklerotische Degeneration der Arterienwand." On the other hand, the arteries in the anterior subarachnoid spaces and their spinal cord branches did not show this proliferative process and narrowing of the lumen, yet the lateral and anterior columns were severely diseased. Anemia, even though transitory, should also have affected the sensitive neurons of the gray matter, which, however, were normal. The marked destruction of the nerve fibers in the periphery of the spinal cord might lead one to assume, with others (Davison and Keschner[2]), a direct action of the syphilitic toxin on the nerve fibers. Perhaps this toxin, circulating in the cerebrospinal fluid, irritated the pia-arachnoid and the wall of the arteries from the outside inward (note the absence of endothelial proliferation) and also penetrated through the outer glial wall and along the pial sheaths of the blood vessels into the spinal cord itself.

1. Alzheimer, A.: Die Colloidentartung der Gehirnrinde, Arch. f. Psychiat. **30**:18, 1897.

2. Davison, C., and Keschner, M.: Myelitic and Myelopathic Lesions: II. Toxic Myelopathy, Arch. Neurol. & Psychiat. **29**:600 (March) 1933.

Other authors described a similar disease of the spinal cord as "myelitis" (Lewandowsky [3]) or as "myelopathia endoarteriitica acuta" (Biernacki [4]). They assumed that interference with circulation following the fibrous degeneration of the walls of the spinal arteries, together with a narrowing of their lumens, was responsible for the destruction of nerve fibers. They failed, however, to account for the normal appearance of the neurons in the presence of the severe disease of the white matter.

DISCUSSION

DR. GEORGE B. HASSIN: What was the condition of the gray matter of the spinal cord? Were there any changes?

DR. ARTHUR WEIL: No.

DR. GEORGE B. HASSIN: How, then, can one explain the flaccid paraplegia? Dr. Weil did not definitely classify the type of changes he found, but merely termed them myelopathy. He demonstrated the presence of infiltrations in some of the blood vessels and also degenerative changes in the white substance of the spinal cord. For some time there was a dispute as to the possible relationship between the degeneration of the nerves and the vascular changes, whether the latter are responsible for the degeneration or vice versa. From his extensive studies of the pathologic process of dementia paralytica, Alzheimer came to the just conclusion that the foregoing types of changes—degenerative and vascular—are independent phenomena which are due to the same cause—syphilis. A toxin of malignant syphilis was evidently also responsible for the analogous changes in the case of Dr. Weil and Dr. Stone. Syphilis of the spinal cord is, I think, a more proper name than syphilitic myelopathy, a term that Dr. Weil evidently imported from New York, where it is much in favor with some neuropathologists. Myelopathy, which means a disease of the spinal cord, is a general term, just as is pathology, which means a science of disease, and does not specify the type of the predominant changes, whether vascular, gummatous, meningeal or degenerative.

DR. PERCIVAL BAILEY: It seems strange that with all the sclerosis in the blood vessels there were no changes in the nerve cells. One would certainly expect to find changes in the anterior horns.

DR. ARTHUR WEIL: The term myelopathy has been used by Aschoff to differentiate the condition from myelitis. The Greek word *pathos* means passive suffering. It indicates, added as the suffix pathy, that a destruction of the tissue takes place which is not followed by a progressive glial reaction or an active mesodermal cellular proliferation. The term myelomalacia (the Greek word *malacia* means disease) is now used by most pathologists as synonymous with softening.

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3. Lewandowsky, M.: Rueckenmarkskrankheiten, in Handbuch der Neurologie, Berlin, Julius Springer, 1911, vol. 2, pt. 1, p. 550.

4. Biernacki, E.: Myelopathia endoarteriitica acuta, Deutsche Ztschr. f. Nervenh. **10**:173, 1897.

Editorial

THE TRAINING OF THE NEUROLOGIST AND THE PSYCHIATRIST

At intervals for over a year articles have been published in the ARCHIVES on the training of the neurologist and the psychiatrist. These were written by four neurologists, two psychiatrists, one pediatrician, one neurosurgeon and one neuropsychiatrist and come from four countries in addition to the United States. The points of view, while geographically varied, were singularly uniform within each specialty, so one must feel that the differences are due not to nationality but to the mental attitude inherent in each specialty.

There seems to be in the minds of some of the writers an idea that neurology and psychiatry are so intrinsically different that a training in one of them renders the physician unfit to appreciate the other. It is difficult to see why this should be. Neither requires such a highly specialized type of mind that its practical mastery should be impossible for any competent physician. Rigid lines of separation between specialties are disappearing everywhere except in large cities. Specialized aid in diagnosis and in therapy should be, and is being, brought to the smaller cities and towns throughout the country. In the changing order, the distinction between the neurologist and the psychiatrist will disappear just as the distinction between the surgeon and the gynecologist is now disappearing.

Neurology and psychiatry are still relatively young specialties, boastful of their emancipation from general medicine. In their development they attained their independence along different lines. Until recent years the neurologist was in private practice and the psychiatrist mainly in institutional practice. To borrow a simile from war, the neurologist has been in the front line and the psychiatrist in the base hospital where he received under his care some of the patients who could not be treated near the front. Conditions of private practice have compelled neurologists to handle psychiatric material, even though they may not have been equipped to do so with complete adequacy. Conditions in hospitals for mental diseases have likewise compelled psychiatrists with little basic training to treat patients with neurologic conditions. But conditions have changed. Psychiatrists are no longer only in the base hospitals; more and more of them are coming up to the front of private practice and, with perfect right, are contesting with the neurologist for cases which were formerly the neurologist's own.

When one reads the articles of this series it is all too patent that the main bone of contention is the large group of the psychoneuroses. Each side claims them as its own. At the present time about 40 per cent of patients seen in private neurologic practice are psychoneurotic, according to the statistics collected by the American Neurological Association. Probably the figure is even larger in private psychiatric practice. Since this is a large slice of each practitioner's daily bread, although undoubtedly reprehensible, it is nevertheless understandable that economic motives may play a rôle in such a controversy.

Nor is that all of the picture. Besides the neurologist and the psychiatrist, many others with less training than they have entered into the competition for practice in the psychoneuroses. Psycho-analysts, some of them without either neurologic or psychiatric training, mental hygienists, child guidance specialists, clinical psychologists, cultists and quacks—"the seven and twenty warring sects of psychotherapy" (Walshe)—now take their place in the front line with the trained physician. Were the neurologist and the psychiatrist each trained to the satisfaction of the other—which is not impossible—and were they more open-minded about learning from "the seven and twenty," they might form a united front in the interests of public health.

How should the neurologist and the psychiatrist be trained? When one reads the articles in this series one gains the impression that there is little controversy between the neurologist and the psychiatrist on this point. Both insist on a thorough basic training in biology and in medicine. For both, adequate laboratory study as a preliminary to and an adjunct of extensive clinical training in the problems of the patient as a whole appears equally important. Thoroughness and breadth of training are equally stressed by both. Again, both deplore the present-day belief of some psycho-analysts that the only qualification for the practice of this form of therapy is that the analyst should himself have been analyzed. Analysis is undoubtedly the best means of learning this special technic and is most helpful in practice, but it never can be a substitute for a knowledge of the nervous system and of psychobiology. The training of Professor Freud, who has not as yet been analyzed, may well stand as a model of what all are aiming for. This is well illustrated by his early study of comparative neurology, with his monograph on the nervous organ of Petromyzon,[1] which was followed by his classic publication on Little's disease.[2] Moreover, he had a thorough psychiatric training. From this background his genius devel-

1. Freud, Sigmund: Ueber Spinalganglien und Rückenmark des Petromyzon, Sitzungsb. d. k. Akad. d. Wissensch. 1878 **78**:81, 1879.

2. Freud, Sigmund: Zur Kenntnis der cerebralen Diplegien des Kindesalters (im Anschlusses an die Littlesche Krankheit), Vienna, Franz Deuticke, 1893.

oped a school of psychology which has contributed much to the understanding of human motives and has given to medicine one of its most useful weapons in psychotherapy. One wonders: Could it have been done otherwise?

The training of the neurologist and the psychiatrist is not a matter of this day alone. More important is the consideration of the future. Only in large cities is narrow specialization possible. Neurology and psychiatry have too much of value to offer to restrict this usefulness to residents of metropolitan areas. The smaller cities, the towns and the country districts need, and should have, neurologic and psychiatric care. Many are the places that could not support both a neurologist and a psychiatrist, to say nothing of a psycho-analyst as well, yet which could support and would welcome a good physician trained in both disciplines. The members of the medical profession will not have fulfilled their duty to their fellow men until they have devoted their energies to the training of such physicians.

The controversy, then, appears to be no real controversy. It is one of method rather than of principle. The principle is one of thoroughness of neurologic and psychiatric training. Dr. Meyer speaks of the need for the "psychiatrically intelligent neurologist." To that one may add "the neurologically intelligent psychiatrist," with the great goal ahead of the medically and surgically intelligent "neuropsychiatrist" who will be the physician to treat patients with nervous and mental disease.

BIRTHDAY CELEBRATION

KARL SCHAFFER

1864-1934

On Sept. 7, 1934, Prof. Karl Schaffer, founder of research on the brain in Hungary, celebrated his seventieth birthday while still enjoying youthful vigor of body and mind. As privat-docent in neurology at the University of Budapest, he became chief of the department of nervous and mental diseases at the infirmary in that city in 1895. Here he established and maintained, with private funds, a histologic laboratory from which he issued numerous important works on Tay-Sachs' disease, tabes, dementia paralytica and other problems in cerebral pathology. Some of his earliest work concerned the mode of invasion by the virus of lyssa, his observations being confirmed and extended some forty years later by Pette, Spielmeyer and Spatz.

Schaffer's interests were strikingly broad. Even as a young assistant he studied the reflex phenomena of hypnotism, the results of this work, especially as regards optic reflex contractions, receiving confirmation thirty-six years later from Metzger. Schaffer contributed largely to the knowledge of the normal anatomy of the brain, reporting observations on the embryology of the convolutional patterns and the anatomic connection between the cerebrum and the cerebellum. In the field of histopathology, in addition to his work on lyssa and on syphilis of the nervous system, he has contributed much on the anatomic basis of hereditary diseases of the nervous system and has created a schema into which can be classed all ectodermogenetic diseases of the nervous system. His crowning accomplishment is a book on the histopathology of hereditary systemic nervous diseases, which appeared in 1925.

Though Schaffer received the title of associate professor in 1901, it was not until 1912 that he became professor, and an institute for research on the brain was erected in his honor in recognition of his services in the sphere of neuropathology. This afforded him greatly enlarged opportunities for research, and he was soon surrounded by enthusiastic students. In 1924, at the age of 60, after some hesitation, he accepted the chair of psychiatry and neurology. Today he is surrounded by many grateful pupils and admirers whose earnest wish is that he may be granted many years of successful activity.

Many honors have been bestowed on Karl Schaffer, but he prizes most his election to honorary membership in the American Neurological Association and the Philadelphia Neurological Society.

Desso Mickolczy, Szeged, Hungary.

News and Comment

AMERICAN BOARD OF PSYCHIATRY AND NEUROLOGY, Inc.

At the annual meetings of the American Medical Association (Section on Nervous and Mental Diseases), the American Psychiatric Association and the American Neurological Association in the spring, 1934, each of the three associations nominated four representatives to a board to be created for the purpose of determining qualifications for recognition of physicians who apply for certification as specialists in the practice of psychiatry and neurology. The nominees for the American Medical Association are: Dr. Walter Freeman, Washington, D. C.; Dr. George W. Hall, Chicago; Dr. L. H. Ziegler, Albany, N. Y., and Dr. J. Allen Jackson, Danville, Pa. For the American Psychiatric Association the nominees are: Dr. Adolf Meyer, Baltimore; Dr. C. Macfie Campbell, Boston; Dr. Clarence O. Cheney, New York, and Dr. Franklin G. Ebaugh, Denver. The following were nominated by the American Neurological Association: Dr. Louis Casamajor, New York; Dr. L. J. Pollock, Chicago; Dr. Edwin G. Zabriskie, New York (appointed to replace the late Dr. Theodore H. Weisenburg, who was nominated originally), and Dr. H. Douglas Singer, Chicago.

Mainly as the result of the efforts of Dr. Clarence O. Cheney, aided by the National Committee for Mental Hygiene, the Board was incorporated on Oct. 19, 1934, under the title, American Board of Psychiatry and Neurology.

On October 20, a meeting of the board was held in New York, at which all the members were present. Officers were elected and by-laws were adopted. The officers are: Dr. H. Douglas Singer, president; Dr. C. Macfie Campbell, vice-president, and Dr. Walter Freeman, secretary and treasurer.

A committee was appointed to formulate forms of application and the methods and procedure to be followed in passing on the qualifications of candidates by examination and otherwise. The report of this committee will be presented at the next meeting of the board, probably at the end of December 1934.

Abstracts from Current Literature

THE BLASTOMATOUS FORM OF DIFFUSE SCLEROSIS OF THE BRAIN (LEUKO-ENCEPHALOPATHIA BLASTOMATOSA). ROBERT PFLEGER, Jahrb. f. Psychiat. u. Neurol. **50**:142, 1933.

A boy, aged 6 years, had mumps in February, measles in March and smallpox in May, 1931. Following these diseases he was found to have strabismus and bilateral ptosis. In June 1931, he became unusually nervous and somewhat deaf. In September 1931, he entered school, but three weeks later was transferred to a school for the deaf. Two days later, while at school, he suddenly became pale and had incontinence of feces. He was sent home; at the end of two days he was apparently well. He returned to school, where he was very unruly. At the end of September, while at play, he was knocked down and fell against an iron hydrant. A physician, soon after the accident, diagnosed the condition as a mild cerebral concussion. On the fourth day after the accident, the boy felt well and played as usual. A few days later, he complained of abdominal pains and had diarrhea for two days. At about this time he began to stutter and his gait became unsteady. In November 1931, he had severe pharyngeal diphtheria with renal involvement. On discharge from the hospital he was unable to stand, and the upper extremities were markedly ataxic; he had also urgency of urination. This condition gradually improved, so that toward the end of December 1931 he could walk with assistance. Within the next few days speech became slurred and slow and fluids regurgitated through the nose; he began to tremble and vomit and had generalized convulsions with loss of consciousness, for which he was admitted to the hospital.

Examination revealed: mental confusion, difficulty in swallowing, some resistance on passive flexion of the head, bilateral signs of involvement of the pyramidal tract, an exaggerated masseter reflex, sluggish pupils, a questionable constriction of the left visual field and normal fundi. The condition became progressively worse; vision became poorer, the optic nerve heads became atrophic, deafness increased and intellectual deterioration was striking. In March 1932, there appeared paralysis of the soft palate; swallowing became impossible, the pharyngeal reflex was absent, and forced laughter set in. He remained in this condition till the end of March, when there were fever, marked spasticity, bronchopneumonia, rigidity of the neck and general exhaustion; he died on March 28, 1932. The cerebrospinal fluid was under somewhat increased pressure and contained 23 cells per cubic millimeter. The mastic curve was 236663. The Wassermann reaction of the blood and spinal fluid was negative.

The clinical diagnosis was diffuse sclerosis of the brain. The anatomic diagnosis was: diffuse cerebral sclerosis, primary tuberculosis of the lower lobe of the left lung, tuberculous lymphadenitis of the peritracheal glands on the left side and bronchopneumonia.

Macroscopic examination of the brain disclosed a pathologic process affecting diffusely both parietal and occipital lobes, with some invasion of the temporal lobes. The most striking gross appearance was the porous and spongy consistency of the brain substance at the point of transition between the temporal and occipital lobes on the left side.

Histologic examination revealed complete disappearance of the myelin sheaths in both occipital lobes, with partial intactness of the fibrae arcuatae and slight demyelination of the lowermost portions of the cortex. The temporal lobes were practically entirely involved in the pathologic process, which was present also in the basal ganglia and optic thalamus, especially the caudal third on the left side; the basal ganglia on the right side were only partially destroyed. There was complete destruction of the callosal fibers in their caudal half, and the cephalic

portion of the corpus callosum showed only partial demyelination. In some areas there were small foci of degeneration of the myelin sheaths; these foci assumed the shape and size of a lentil and seemed to have a perivascular arrangement. The pathologic process was less marked in the cephalic portion of the temporal lobe, and the most anterior portions of the frontal lobes appeared normal. The pyramidal tracts in the internal capsule showed marked degeneration, which could be traced to the lowermost portion of the cord, more on one side than on the other.

In finer detail, histologic study revealed evidences of acute lesions interspersed between chronic lesions. In the former there were massive accumulations of various forms of cells, large plasma cells and round, sharply outlined polygonal cells with or without prolongations; here and there were cells containing several nuclei and others with imperfect nuclear divisions. Between these larger cells were numerous small cells resembling lymphocytes, which with Hortega stains appeared to be microglia cells. At the margin of the cortex, at the periphery of the lesion, were numerous typical fat-containing granular cells. In some areas these elements had a palisade-like arrangement, which was most marked in the left temporal lobe. The axis-cylinders, though pathologic, were fairly well intact, particularly in areas in which the cellular mass was free from degeneration. It is noteworthy, however, that glia fiber formation was of the same intensity in the acute as in the chronic lesions. In some places there was a sharp line of demarcation between the acute and the chronic foci; in others the transition was gradual. The chronic lesions consisted of glia fibers, with spindle cells, some of which were polynuclear while others contained atypical nuclear formations. The scar formation was not uniform; in some areas the fibers appeared club-shaped; in others they were more delicate, and in still others they were wide, bow-shaped and wormlike.

The vascular reaction was in marked contrast to the changes in the walls of the vessels ordinarily observed in diffuse sclerosis. There was no uniformity in the cellular reaction in the vascular walls. Five different cellular types of infiltrations were present: (1) lymphoid cells with plasma cells; (2) large plasma cells with almost no scavenger cells; (3) exclusively fatty granular cells; (4) small cellular elements that were definitely not lymphocytic, and (5) mixed and transitional forms. These changes were also observed in the vessels in the sclerotic areas. The ectodermal invasion into the perivascular mesodermal territory was also striking, as was the pericapillary cuffing in the pyramidal tract throughout the pons, medulla oblongata and spinal cord.

On the basis of the histologic observations in this case, Pfleger believes that diffuse sclerosis may exist in four forms: (1) inflammatory, (2) degenerative, (3) heredodegenerative and (4) blastomatous. He regards this case as a blastomatous form, with primary fiber formation which assumes the character of a system disease affecting principally the corticospinal tracts in the brain and cord.

KESCHNER, New York.

COURSE AND LATE SEQUELAE OF INJURIES TO THE SKULL AND BRAIN. R. BRUN, Schweiz. Arch. f. Neurol. u. Psychiat. **31**:182, 1933.

This contribution contains such a wealth of material—there are 328 references—that much of it, particularly the portion dealing with the immediate results of injury to the head, cannot be adequately reviewed. Brun expresses the view that a protracted traumatic delirium is something more than a mere continuation of the twilight state with motor unrest which often appears after the initial coma or immediately following the injury, since delirium may supervene some time later without preliminary disturbance of consciousness. Patients with symptoms closely resembling the Korsakoff psychosis are separated from the main group because most of them, in Brun's experience, are alcoholic. The prognosis is bad in all cases with delirium because of the likelihood of permanent mental impairment.

Although an abscess of the brain of traumatic origin may first manifest itself clinically years after the trauma, so-called late meningitis is, as a rule, attributable to a previous injury only in the presence of infection in the accessory sinuses, middle ear or labyrinth and a demonstrable communication between these structures and the cranial cavity. Neurologic signs as well as mental symptoms of gross injury to the brain need not necessarily have existed from the time of injury, nor is a progressive increase in their severity precluded. Traumatic epilepsy, which in most cases appears within seven weeks of the injury, frequently follows injury to the parietal lobe. While the existence of traumatic parkinsonism can no longer be doubted, cases falling in this group are generally not cleancut, as the injury is seldom confined to the extrapyramidal system. The older belief that the basal ganglia were rarely involved in traumatic injuries must, in the light of recent work, be revised. Trauma as a predisposing or aggravating factor in tumor or other chronic diseases of the central nervous system is in general of doubtful significance.

The organic and functional elements in posttraumatic cases are often inextricably mingled, and psychogenic symptoms may be due to preexisting complexes rather than to any desire for compensation. In support of his plea for careful objective examination, Brun cites instances in which autopsies on persons supposedly neurotic have revealed gross cerebral damage. Modern refinements in the examination of the cerebrospinal fluid and tests for its rate of resorption are highly technical and difficult of interpretation. Encephalography, though often helpful when its results are interpreted in the light of other findings, has not justified the enthusiasm of its early advocates. Brun stresses the importance of careful and repeated search during the neurologic examination for "microsymptoms" or slight deviations from the normal, which, in his experience, are seldom if ever lacking in the presence of structural damage to the brain. In view of the difference of opinion relative to the importance of these findings, Naegeli's concept of traumatic encephalosis represents a real advance. The condition is characterized, in addition to the headaches, vertigo and other symptoms common to the posttraumatic state, by a definite impairment of the mental capacities. This concept is not well established, for there are all gradations between the slightest degrees of so-called traumatic neurasthenia and the most severe forms of traumatic dementia. Traumatic encephalosis can and should be differentiated, however, from premature arteriosclerotic and senile dementia, which may follow injury to the head in older persons.

Traumatic encephalosis, in Brun's experience, consists not only in a quantitative but in a qualitative impairment of attentiveness, comprehension and other elementary functions as well as of the higher faculties. Disturbed behavior and even permanent changes in character are often associated with these intellectual defects. Although dulling of affect, amounting at times to aspontaneity, is common, a few of the author's patients showed a hypomanic reaction with pronounced euphoria and lack of insight. Using the foregoing criteria, Brun classified 56 of his 175 cases of injury to the head as examples of traumatic encephalosis. All of the 56 patients showed more or less definite objective evidence, slight deviations from the normal for the most part, of a previous cerebral contusion. Eighteen had a "frontal lobe ataxia" of the contralateral leg; 7 presented amyostatic symptoms, and 3 had traumatic epilepsy. A preliminary loss of consciousness was not recorded in all of the mild and moderately severe cases, but in half of the severe cases the patients had been unconscious longer than twenty-four hours. Some patients who had been unconscious for days, on the other hand, showed no permanent mental defect. On the basis of his own experience and that of others Brun concludes that traumatic encephalosis is essentially a syndrome of the frontal lobe; that when the traumatic force is not applied in a direction corresponding to the sagittal axis of the skull and does not injure the frontal lobe subsequent development of an encephalosis is unlikely.

The prognosis in these cases is always uncertain but not necessarily bad, particularly in young persons, although years may be required for complete

recovery. In older patients, traumatic encephalosis may progress to complete dementia. In general, the degree of disability is greater among the highly trained, and the patients are always mentally and physically less productive than they were before the injury.

Forty-nine patients of the entire series showed secondary neurotic symptoms, but of the 14 in this group who in addition seemed to be suffering from traumatic encephalosis, the latter condition had attained a high degree of severity in only 1. Of 24 patients suffering from traumatic neurasthenia or visceral neurosis, a condition attributed by the author to injury of the vegetative centers, signs of a cerebral contusion were detected in only 10, and none showed qualitative intellectual impairment. The fright attending an injury may likewise produce a vegetative imbalance (*Schreckneurose*), but, as in the visceral neurosis and contrary to what occurs in a secondary psychoneurosis, symptoms appear immediately after the trauma and are not psychogenic in the sense of a wish fulfilment.

DANIELS, Denver.

CEREBELLAR DEMENTIA PARALYTICA. MAX BIELSCHOWSKY and ROBERT HIRSCHFELD, J. f. Psychol. u. Neurol. **45**:185, 1933.

The authors review the literature and comment on the rarity of cerebellar dementia paralytica. The case they report is that of a man, aged 45, who was admitted to the Charité in Berlin with vertigo, reeling gait, clumsiness of both upper extremities and difficult speech. For a number of weeks prior to admission, he had had defective memory, periods of depression, increased irritability and slight confusion. Examination at the hospital disclosed: vertical and horizontal nystagmus, more marked on looking to the extreme right; pupils of somewhat irregular contour, the right being a little wider than the left though both reacted normally to all stimuli; normal fundi; dysarthria; hypotonia of the right upper extremity; bilateral dysdiadokokinesis and ataxia on performing the finger-to-nose test; normal tonus in the lower extremities; active and equal tendon reflexes; no pathologic reflexes; active superficial reflexes; a positive Romberg sign; slight falling to the right; broad-based ataxic gait, and definite diminution in the speed of all volitional movements. Mental examination revealed no abnormalities. The patient remained in the hospital for three weeks, during which the vertigo disappeared, but the clumsiness and unsteady station and gait persisted. The clinical diagnosis was multiple sclerosis.

During the next six months, speech and gait became worse, so that the patient became chair-ridden; soon thereafter the clumsiness in the upper extremities became so marked that he was unable to feed himself; there also developed at this time disturbances in control of the bladder and rectum. He remained in this condition during the next four years, when he suddenly grew worse. Tremor of the head and limbs appeared; speech became almost impossible; chewing and swallowing became extremely difficult, and the sphincteric disturbances were more marked; neither forced laughter nor spontaneous crying was observed. Examination at this time revealed: poor nutrition with general muscular wasting; a masklike facies and a coated fibrillating tongue, general muscular rigidity and an irregular right pupil. Both pupils reacted well to light. Owing to immobility of the eyeballs, convergence could not be tested. Winking occurred rarely. In spite of the loss of power of fixation, there was no palsy of the extra-ocular muscles. The patellar and ankle reflexes as well as the abdominal reflexes were not elicited, but the periosteal reflexes were exaggerated in the upper extremities. There were no pathologic reflexes; the corneal reflexes were absent. Sensation to pin prick was felt on both sides of the body. The fundi oculorum showed no abnormalities. The spinal fluid gave a weakly positive reaction to the Pandy and Nonne-Appelt tests and a negative reaction to the Wassermann test. The gum mastic curve approximated that obtained in tabes. Tests for liver function yielded no conclusive results. The blood sugar content was strikingly elevated. The rigidity

gradually became more marked, the severity of all symptoms and signs increased, pneumonia supervened and death occurred at the age of 51, six years after the onset of the illness.

Necropsy revealed brown atrophy of the liver and spleen.

Gross examination of the brain revealed no abnormalities of the convolutional configuration; the pia over the orbital surface of the frontal lobes and over the first and second temporal convolutions appeared dull but could be readily detached from the brain.

The most noteworthy changes were those in the cerebellum, which was reduced to less than one third its normal volume. Corresponding to this reduction in size, the individual lamellae of the ventral and dorsal surfaces of the cerebellar hemispheres as well as of the vermis were narrow. The crura cerebelli ad pontem were flattened and depressed. The frontal convolutions were somewhat deeper than normal. The basal cerebral nerves appeared normal and the olives were more prominent than usual.

In preparations stained for glia the cortex and white matter of the cerebellum appeared markedly sclerosed, though not sufficiently to obliterate completely the gross structure. Histologic examination left no doubt that the pathologic process was dementia paralytica. The leptomeninges and vessels of the white substance showed an abundance of lymphocytic and plasmocytic infiltration. Similar alterations were observed in the cerebrum and other parts of the brain. The olives and the olivocerebellar and pontocerebellar systems, though severely affected, showed an entirely different histologic picture from that observed in olivopontocerebellar atrophy. It is also noteworthy that there were a large number of small miliary gummas and glial foci with an enormous overproduction of iron-containing rod cells; this is emphasized because these alterations are regarded as conclusive evidence that the process was not one of hereditary dementia paralytica.

KESCHNER, New York.

PATHOGENESIS AND TREATMENT OF TABETIC OPTIC ATROPHY. SCHIFF-WERTHEIMER and LHERMITTE, Ann. d'ocul. **170**:165 (Feb.) 1933.

Study of the pathogenesis of tabetic optic atrophy depends on clinical, anatomic and biologic facts. From a clinical point of view the authors summarize briefly the classic conceptions: Early contraction of the peripheral visual field, disturbance of retinal adaptation and the ophthalmoscopic appearance of simple atrophy, without symptoms of inflammation of the optic nerve, mark the beginning of visual disturbances. They point out the rarity of central scotoma, the normal appearance of retinal vessels until the disease is in an advanced stage and the relative frequency of mental disturbances in tabetic patients who suffer from optic atrophy.

The pathologic study of tabetic optic atrophy shows lesions which involve the myelin sheaths, the neuroglia and the vascular connective tissue and lesions which vary according to the rapidity of the evolution of the disease and the duration and stage of the process when the examination is made. The myelin disappears progressively, being phagocytosed by myelophages and myeloclasts, which in turn are absorbed by the blood vessels. The axis-cylinders become fragmented before they are phagocytosed. The neuroglia proliferates sometimes in the form of phagocytic elements and at other times in the form of fibrillae which secondarily produce a true sclerosis of the nerve, especially in the retrobulbar region. Meningovascular lesions vary in intensity and are almost constant, but are not proportionate to the changes affecting the axons. The authors did not find in their cases the constant and early perivascular lymphocytic infiltration described by Pierre Marie and Léri, who considered tabetic optic atrophy as an interstitial neuritis and its primary lesion as a meningovascular inflammation. The topography of the changes in the optic nerve shows that they are diffuse and occur mostly in the retrobulbar region of the nerve, and that the secondary degenerations are slight.

Both biologic and anatomic considerations indicate that the degeneration of the nerve fibers is not necessarily closely connected with the meningovascular lesions. Recalling the investigations of Igersheimer, the authors insist that the infectious agent is absent in the interior of the nerve. The pathogenesis of tabetic optic atrophy has not been entirely elucidated any more than has that of the disease of Duchenne. Schiff-Wertheimer and Lhermitte have made a critical study of the diverse pathogenic conceptions of tabes dorsalis and conclude that they have still to discover the true cause of the characteristic lesions of the nerves in this disease in general and of the accompanying complications of the optic nerve. These lesions cannot be explained by simple mechanisms, and they make possible the conception of a toxic origin proposed in certain recent works.

Uncertainty as to the pathogenesis causes doubts as to the therapeutic procedure. The evolution, which may be slow or rapid, but which almost always destroys the optic nerve, has encouraged most daring therapeutic experiments. Antisyphilitic treatment is often ineffective, regardless of the manner in which chemotherapy is used; it has no active or especially dangerous effect on the optic nerve fibers, provided the medication is given in careful doses during a prolonged period. Treatment by the subarachnoid route, whether an injection of air, of neoarsphenamine or of mercury, as well as cerebrospinal drainage, often seems to be a useful adjuvant of the usual chemotherapeutic treatment, especially in the early stages of the infection.

Schiff-Wertheimer and Lhermitte are seeking to determine the results obtained in the treatment of tabetic atrophy by malariatherapy. The results vary, depending on the stage of the disease at the beginning of the treatment. Apparently the action of malariatherapy is more uncertain in the treatment for tabes and for the accompanying complication of the optic nerve than it is in dementia paralytica; nevertheless, successful results have been obtained by malariatherapy, as described by Horn and Kauders. The various forms of pyretotherapy, especially sulphurated oil, are preferred to infection therapy. The authors briefly review other methods of treatment (electropyrexia, the medication called "vasodilator") and suggest a surgical treatment which liberates the optic nerves from their meningeal sheaths. Although these therapeutic experiments have not been successful in many cases, they have assisted in the stabilization of infection. The authors conclude that this form of infection should be treated cautiously and for a long period in spite of visual disturbances which may arise during the treatment. These disturbances cannot always be ascribed to the treatment.

BERENS, New York.

FIELDS OF VISION IN INTRACRANIAL LESIONS. H. M. TRAQUAR, Brit. M. J. **2**:229 (Aug. 5) 1933.

Modern perimetry includes, in addition to the separation of blind from seeing areas, an estimation of the functional efficiency of the whole field. The intensity of a defect means the degree of blindness present and the uniformity and the relative distribution of the blindness within its area. By the margins of a defect is meant the nature of the edges, whether abrupt or sloping. Under normal conditions, the fields for a colored object and a white object of the same size preserve a certain relationship in regard to area. When this is present in pathologic conditions, as for example in concentric contraction of the field, proportion is said to be present. In certain abnormal conditions, the field for the red object may be unduly small in proportion to the amount of reduction for white; this is spoken of as disproportion. Congruous defects are simultaneous in appearance, coextensive and equal in intensity in the two fields. Congruity or incongruity has a localizing value, since all hemianopias due to suprageniculate lesions must be congruous. There is one exception to this rule. Owing to the fact that the temporal and nasal portions of the field are not normally coextensive, the temporal field being the larger by a crescentic area about 30 degrees wide at its widest part, this area corresponds to the nasal periphery of the retina, and the nerve fibers concerned have no mates. There are no corresponding temporal retinal areas. It is possible, therefore, in a suprageniculate hemianopia for the temporal 30 degrees of the

binocular field to be affected alone, or for this part to be spared in an otherwise congruous homonymous hemianopia.

Once the visual fields have been determined, their interpretation may give anatomic or pathologic data as to the lesion and may aid a great deal in prognosis and treatment. It is emphasized, however, that diagnosis should never depend on perimetric findings alone.

On tracing the visual pathway backward from the optic foramen, it is found that different forms of field defect are produced at different levels. The pathway may be regarded as consisting of a suprageniculate portion, including the cortex and the radiations, and a subgeniculate portion, which is divided into subchiasmal, chiasmal and suprachiasmal sections. An accompanying table shows the relationship between the different parts of the visual field and the more commonly occurring lesions and the field changes produced thereby. The table is unusual in its comprehensiveness and clarity.

It has hitherto been believed that the visual fibers on leaving the external geniculate body pass directly backward in a defined band until they spread out to their distribution in the calcarine area of the occipital cortex, the internal fibers passing to the anterior part of the cortex, the more external fibers to the posterior part. In recent years, however, a different view has been advocated, according to which the fibers of the radiation, on leaving the geniculate body, divide into two main groups: dorsal and ventral. Only the dorsal fibers pass directly backward. The ventral bundle is said to pass downward and forward into the temporal lobe, extending, in a fan-shaped arrangement, as far forward as the tip of the anterior horn of the lateral ventricle, which it surrounds before turning backward to the lower part of the visual area of the occipital cortex. This arrangement is known as Meyer's temporal loop. If this is so, lesions in the anterior part of the temporal lobe should produce superior homonymous quadrantal defects in the fields, and the defects in the two fields should be congruous. Clinical evidence tends to contradict this and to deny the existence of Meyer's loop.

Changes in the visual fields in cases of tumor of the temporal lobe differ greatly from those in tumors of the occipital region in the frequency of incongruity and of upper quadrantal defects, and the relative rarity of sparing of the central area. These findings suggest to the author considerable support for the view that it is the optic tract and not the optic radiation which is affected by tumors of the temporal lobe.

FERGUSON, Niagara Falls, N. Y.

PHENOMENA OF PREHENSION AND MOTOR NEGATIVISM AS SYMPTOMS OF FOCAL CEREBRAL LESIONS. S. GULLOTTA, Riv. di pat. nerv. **41**:225 (March-April) 1933.

The author discusses the grasping reflex phenomena of prehension and motor negativism from the clinical standpoint and reports the clinical history and pathologic changes in four cases in which forced grasping was clinically detected. He thinks that the presence of the phenomena of prehension and motor negativism is more frequent than is suspected. In the cases studied clinically, there was predominance of forced prehension or forced grasping on one side. In these cases the grasping was voluntarily resolved. In addition, in four of five cases in which the phenomena were studied, a sort of attraction toward the stimulus was detectable. Such a tendency to prehension manifested itself as a reaction of "nearing" and "possession." These reactions were always accompanied by localized motor negativism, slight hypertonia and slight involvement of the pyramidal tract.

In all cases, in addition to internal hydrocephalus, lesions of the neostriatum and thalamus were always present, and were more pronounced on one side. Only one case disclosed the presence of a hemorrhagic focus in the frontal area. This hemorrhage took place, however, after the phenomena of prehension were registered clinically.

Irrespective of the phenomena of prehension that may appear as an expression of temporary isolation (during sleep, in the agonal period, in the course of strong

emotion and particularly in the case of severe pain), the phenomena of prehension have an importance in pathologic conditions of the nervous system. When present bilaterally and not constantly they are an indication of internal hydrocephalus; when they appear to be circumscribed or predominant on one side they have a localizing value. If they appear as forced grasping, not solvable voluntarily, even through painful stimuli, they stand for a contralateral frontal lesion, particularly if signs of tumor of the brain are detected. If the forced grasping is voluntarily resolved, if it is not the result of painful stimuli and if it is accompanied by motor negativism, the grasping then indicates lesions in the contralateral neostriatum and thalamus, particularly if developed in the course of arteriosclerosis.

The fact that the appearance of grasping phenomena follows lesions of the basal ganglia demonstrates, according to Gullotta, the existence, in addition to the cortical areas, of other areas which normally inhibit grasping movements. He thinks that there is an inhibitory system which runs from the cortex down to a center of the reflex of prehension or grasping reflex, localized in all probability in the cervical cord. A lesion involving the inhibitory system at any one of the cortical, striatal, thalamic or mesencephalic levels would result in the appearance of phenomena of prehension.

FERRARO, New York.

HOMOSEXUALITY IN WOMEN. HELENE DEUTSCH, Internat. J. Psycho-Analysis **14**:34, 1933.

Deutsch reports her findings in eleven homosexual women who were analyzed completely. None of the patients exhibited signs of masculinity in physical appearance or body build. Anatomically and physiologically they could be described as feminine. Analysis in these cases shows that, as children, little girls give up a desire for a penis by wishing for a child. The harder it is to endure frustration of the desire for a penis, the more aggressive will be her reaction to the frustration of her desire for a child. It is curious that the most feminine wish for a child occurs in women in whom the psychic struggle of the castration complex or penis envy is most severe. The early suppression of masturbation by the mother results in a strong feeling of hate toward the mother. This causes a strong feeling of guilt, which is transformed into a masochistic attitude. In overt homosexuality in women the relation is one of alternating mother and child situation, in which sometimes one and sometimes the other plays the rôle of a mother. When the wishes for a penis and a child have to be given up, and if the father for one reason or another repulses the girl, she has to flee back to the place where she enjoyed in the past the greatest comfort, love and security, that is, to her mother, even though she has felt at one time a hate for her. The hate is caused by the fact that the child holds the mother responsible for the lack of a penis. The danger point lies in the rejection by the father, which causes a bisexual oscillation between father and mother and may result in a neurosis, heterosexuality or inversion. One of the worst results of oscillation is in the arrest between the two in a state of persisting narcissism. This represents an arrest of a special kind in the swing of the libidinal pendulum between the mother, who is both hated and loved, and the father, who rejects the child. Fixation on the mother is really a regression, occurring when the girl finds that her father does not want her. This retrogression to an early pregenital level also causes the resurrection of the old penis envy, and the result is the development of a distinct homosexual type which denies the lack of a penis. These patients choose love objects who confirm their masculinity. Female homosexuality cannot be explained by mere primal fixation on the mother; what happens is that the oedipus complex is not resolved and the child goes back to its early mother fixation. Of course there are cases in which the libido has known only one object, that is, the mother. Such cases, however, are nothing else than cases of general psychic infantilism, with diffused anxieties and perversions. Although the swing to homosexuality is determined at puberty, the foundation is laid in early infancy.

KASANIN, Howard, R. I.

RELATIVE MERITS OF SPINAL AND ETHER ANESTHESIA. H. L. FOSS and L. J. SCHWALM, J. A. M. A. **101**:1711 (Nov. 25) 1933.

Foss and Schwalm studied the mortality figures, based on all deaths in the hospital following 4,000 consecutive major operations, one-half performed under spinal anesthesia and one-half under ether. Deaths in the operating room in this series were more common with ether than with spinal anesthesia, in a proportion of 10:1. Postoperative deaths occurring in the first few days following operation are not more common after spinal anesthesia. The ultimate death rate in the hospital is practically the same following the administration of the two anesthetics. In this series of 4,000 closely parallel operations the difference was only 0.15 per cent. While pulmonary complications are no less common following spinal anesthesia, there is not a noteworthy increase in the incidence of pulmonary deaths when this anesthetic is used over those occurring following the administration of ether. Spinal anesthesia can be used more or less as a routine measure in operations below the diaphragm. It is the anesthetic par excellence in most acute abdominal emergencies, acute appendicitis, intestinal obstruction and perforated ulcer. There is some evidence to suggest that certain changes in the spinal cord may follow the injection of procaine hydrochloride into the subarachnoid space, but we are yet to learn whether or not this is common, permanent or of any especial significance, provided an aseptic and proper technic is observed. Improvements in technic have rendered the use of spinal anesthesia as safe as the use of ether anesthesia, and, in most instances, it is infinitely more convenient and helpful and is followed by fewer complications. The majority of patients prefer spinal anesthesia. The authors agree with Pemberton that they have never seen a serious complication due to the administration of spinal anesthesia.

EDITOR'S ABSTRACT.

MURDER AS AN EXPRESSION OF NORMAL EMOTIONAL STATES. H. HELWEG, Acta psychiat. et neurol. **7**:201, 1932.

The author reports several cases to illustrate his thesis that murder may be an expression of a normal emotional reaction accentuated by concomitant circumstances rather than a definitely pathologic act. The first example, that of a man of low intelligence who murdered the children of his mistress in revenge for her desertion of him, illustrates the type of person of low intelligence whose emotional life—usually only slightly inhibited—completely controls him, particularly when excited by the actions of other people. The second example, that of a man who killed his wife because she would neither accompany him nor allow him to emigrate as a way out of his financial troubles, illustrates that a normal person of a not aggressive type and inclined to have rigid ideas may be placed in unhappy and distressing circumstances from which he sees no escape except by the murder of some person who is an obstacle to any relief from the situation. The third example, that of an old, poverty stricken, infirm man who killed his aged and insane wife because he felt she was being ill treated in the hospital and would be happier dead, illustrates a situation similar to the last. He believed that his wife was unhappy; he could do nothing to help her and murdered her as a solution for the distressing and otherwise unalterable situation. The fourth example, that of a young man who killed one woman and wounded another because the first made fun of him, perhaps an illustration of the psychopathic personality (namely, that of a person who has acquired little ethical sense and reacts violently and often asocially to minor irritations). The fifth example is that of a young man who killed his mistress because she excited him sexually to an inordinate degree and then through coquetry and capriciousness refused to satisfy him.

PEARSON, Philadelphia.

PSYCHOANALYTIC ASPECTS OF SUICIDE. KARL A. MENNINGER, Internat. J. Psycho-Analysis **14**:376, 1933.

The individual man in a measure always creates his environment, and thus the suicidal person must help to create the very thing from which in suicide he takes flight. If suicide is explained dynamically, one is compelled to seek an explanation for the wish to put oneself in a predicament from which one cannot escape except by suicide. Suicide is a peculiar kind of death which entails three elements: the element of dying, the element of killing and the element of being killed. Each element requires separate analysis. Each is an act for which there exist motives, unconscious and conscious. The various unconscious mechanisms in the three elements are discussed by the author with the stress on the ego, the superego and the id. Suicide is often a self-punishment for sadistic wishes, with the retribution in this extreme form of masochism. Taking up the problem of suicide in families in which more than one member has committed suicide, the author states that unconscious death wishes are often projected against the members of the person's own family. When the member of the family dies or kills himself, this produces a terrific feeling of guilt, and the patient then punishes himself by inflicting death on himself. Sometimes this is done by dreams of being executed, hanged, killed or sentenced to life imprisonment. It is interesting that men more frequently choose shooting and women drowning or the taking of poison or gas. These methods are obviously clearly related to the masculine and feminine rôles in sexual life. There is also partial suicide, by which the author means certain neurotic syndromes and neurotic character manifestations which may be focalized. Certain conversion hysterias are focal, self-destructive attacks, while aceticism and martyrdom represent chronic generalized self-destruction.

KASANIN, Howard, R. I.

THE TRIAL OF GUITEAU. FREDERICK A. FENNING, Am. J. Psychiat. **13**:127 (July) 1933.

Charles Guiteau, tried in 1882 for the murder of President Garfield, set up the defense of insanity. Fenning reviews the expert medical testimony for and against Guiteau and speculates as to the outcome of the trial were it held today. The hearing was held before the days of Freud, Ehrlich or Kraepelin. The defendant testified that he was perfectly sane until four days before the shooting, when he became the agent of God, inspired to carry out divine instructions by removing the President. He stated that having carried out these instructions he returned to normal sanity. Dr. Edward Spitzka, testifying on his behalf, stated that Guiteau had been in a "morbid state" all his life and that he was insane at the time of the crime and at the time of the examination. The government experts were skeptical of "moral insanity," contending that this phrase enjoyed no official medical recognition but was, in effect, synonymous with viciousness. They pointed out that the impulse had been successfully resisted for a time and could not, therefore, be an irresistible impulse. Dr. John P. Gray, then editor of the *American Journal of Insanity*, testified that moral insanity was merely a loose term designating wickedness. The boastfulness which characterized the defendant's behavior was considered by Dr. Gray and Dr. Fordyce Barker (witnesses for the prosecution) as a result of sane conceit and love of notoriety and not as a product of disease; it was termed a vice rather than a psychosis. Justice Cox, in charging the jury, stated that "a man cannot escape responsibility by baptizing his own deliberate resolves with the name of inspiration." Guiteau's conviction was sustained by the United States Supreme Court.

DAVIDSON, Newark, N. J.

THE SO-CALLED LAW OF ANTICIPATION IN MENTAL DISEASE. ARTHUR S. PATTERSON, J. Neurol. & Psychopath. **13**:193 (Jan.) 1933.

The belief that mental disease tends to appear at an earlier age in each succeeding generation has been designated, chiefly by Morel and Mott, as the law of anticipation. The present study of this law is based on over 4,000 cases

taken from the London County Mental Hospitals, material that formed the basis of Mott's conclusions. Patterson criticizes the manner in which Mott's material was collected and its nature. The comparatively short age period between parent and child (average 13.7 years) favors the appearance of spurious antedating in the children's generation. This is exemplified by examination of some of Mott's patients during the next twenty years, which showed a marked reduction in the degree of anticipation, which is to be expected from the fact that parents are on the average thirty years older than their children. One must permit the child to live past the danger age of mental disease if one is to have accurate comparative data.

The fact of parenthood itself presupposes a probable age of 25 or 30 before affliction with mental disease, which increases the percentage of psychotic parents in later life. The occurrence of certain common mental diseases, e. g., schizophrenia, in early adult life diminishes the frequency of mental disease in parents in the earlier age periods, as there is less chance of becoming a parent. The statistics are likewise influenced by the earlier recognition of mental disease in the offspring of the psychotic, as well as the tendency to earlier commitment nowadays.

The author concludes that the law of anticipation is unproved and that conclusions of eugenic or prognostic import based on it are unwarranted.

SPERLING, Philadelphia.

THE INFLUENCE OF POSTERIOR PITUITARY HORMONE ON THE ABSORPTION AND DISTRIBUTION OF WATER IN MAN. F. H. SMIRK, J. Physiol. **78**:147 (May 23) 1933.

When diuresis is prevented by administration of the hormone of the posterior lobe of the pituitary gland (from 0.05 to 0.1 cc. of pitressin) the absorption of 1 liter of water takes place normally. The extra water causes an increase in the weight of the leg, which weight is maintained at about its maximum level for a longer period than in experiments in which diuresis is allowed to take place normally. The degree of general dilution of the blood is at first usually much greater than would be expected from partition of water among the water-bearing tissues of the body; and whereas the degree of dilution of the blood was found to average 7 per cent, the degree of dilution in the tissues of the leg averaged about 1.3 per cent. The spontaneous reconcentration of blood which was diluted as a result of giving pitressin and water is due to the continued transference of water from the blood to the tissues, lymph or serous cavities at a time when absorption is needed.

In man the absence of diuresis after the administration of pitressin cannot be attributed either to a delayed absorption of water or to the excessive removal of water from the blood by the tissues; nor can it be attributed to general delay in the passage of water from the blood to the tissues, since there is clear evidence of an excess of water in the tissues.

ALPERS, Philadelphia.

TUMOR OF THE PARIETAL LOBE SHOWING JACKSONIAN SENSORY SEIZURES. ALEXANDER SILVERSTEIN, J. Nerv. & Ment. Dis. **77**:371 (April) 1933.

The case of a man, aged 42, with a glioma of the parietal lobe is described in detail. There had been two or three attacks of jerking of the left side of the face, affecting the speech. There was a progressive increase both in frequency and severity of jacksonian sensory and motor seizures affecting the larynx, pharynx and tongue and spreading to the face, which were accompanied by a mild left lower facial paresis and pointed to an irritative lesion of the right parietomotor cortex. A Horner syndrome on the right side suggested that the left facial weakness might be the beginning of a peripheral facial palsy. With the development of atrophy of the left hand, the diagnosis was assured, and an operation was undertaken. The tumor extended from the sylvian fissure half way to the vertex, and from the rolandic fissure to the postcentral convolution. It was firm and cartilaginous. Pathologic examination revealed a spongioblastoma multiforme.

Silverstein comments on the rarity of jacksonian fits affecting both extremities simultaneously. The most important feature of the case was the atrophy of the left upper extremity, which was noted early and gradually affected the whole left upper extremity despite the first postoperative improvement. Atrophy of the uvula and tongue and wasting of the left side of the face were later developments.

HART, Greenwich, Conn.

DEMONSTRATION OF COUNTERDEVIATION OF THE EYES BY MEANS OF STRUYCKEN'S APPARATUS. R. CAUSSÉ, Rev. d'oto-neuro-opht. **11**:88 (Feb.) 1933.

Counterdeviation of the eyes during lateral rotation of the head was observed over a century ago, but its true cause was not understood until after the experiments and investigations of Breuer. Only after Versteegh's investigations in 1928 was it proved that the position of the eyes in relation to the different positions of the head is controlled by the utricular macule. Maxwell, Tait and McNally, Bárány and de Nó developed this subject still further. Various investigators found various results as to the amount of counterdeviation, and Caussé thinks that these discrepancies are due to imperfect methods of observation and to imperfect instruments. The advantages of the Struycken apparatus are: The telescope is fixed to the patient's head, observation is made of the part of the cornea most distant from the center of rotation, and the landmarks used are corneal vessels on both the nasal and the temporal sides. The last eliminates the error from slight movements of elevation and lowering of the globes, since in movements of counterdeviation the vessels move upward on one side and downward on the other side. A scale for measurement of the degrees of counterdeviation is interposed in the instrument, and a goniometer is attached to measure the degree of rotation of the head.

DENNIS, Colorado Springs, Colo.

FIRST CEREBELLAR SIGNS IN TUMORS OF THE CEREBELLOPONTILE ANGLE. HETEROLATERAL CEREBELLAR SYNDROME WITH EXTRAPYRAMIDAL PARKINSONISM DUE TO CEREBELLAR OVERFUNCTION. K. HENNER, Rev. neurol. **2**:377 (Sept.) 1933.

In a series of twenty-one proved and suspected cases of tumor of the cerebellopontile angle, Henner encountered the following peculiar syndrome on four different occasions: Whereas on the side involved there was usually considerable dyssynergia with other common cerebellar signs, on the opposite side was some indication of catalepsy, as well as sudden arrest of the rebound phenomenon, limitation of the swing following the patellar reflex and sluggish and rather rigid voluntary movements of the hand. In certain cases even plasticity and hypometria have been found on the opposite side, with increase of the simple postural reflexes. But in no case was there the actual cogwheel phenomena. In six other cases one or more of the preceding symptoms were present in addition to the classic cerebellar signs on the side of the tumor. Henner draws the conclusion that the parkinsonian syndrome may be due to overfunction of the cerebellum, which now lacks its normal restraint. Certain other examples of hyperfunction of the cerebellum leading to rigidity and akinesia have been reported, and in some instances a primary overfunction of the cerebellum may apparently be succeeded by characteristic hypofunction with the classic cerebellar signs.

FREEMAN, Washington, D. C.

A NEW MODIFICATION FOR PROTEIN DETERMINATION IN THE CEREBROSPINAL FLUID. H. STEFAN, Arch. f. Psychiat. **99**:317 (March) 1933.

The method reported is designed for the determination of the total protein and globulin, and the ratio of the globulin to the albumin. The total protein is determined by precipitation through the aid of nitric acid. The spinal fluid is diluted

with 4 parts of physiologic solution of sodium chloride, and this is further diluted with the addition of calibrated quantities of saline solution to form dilutions of spinal fluid in saline solution ranging from 1:5 to 1:90 in nineteen tubes. The nitric acid is added to these solutions, and in precipitating the proteins a ring is formed between the nitric acid and the spinal fluid solution. The amount of protein present in the spinal fluid will determine the concentration in which such a ring is obtained. As the dilution becomes weaker, more protein is necessary to obtain a positive reaction. A chart is appended which shows the amount of protein necessary to form rings in each of the dilutions, which was computed on the basis of standard protein solutions. Sixteen milligrams per hundred cubic centimeters will give a ring in a dilution of 1:5, whereas in a dilution of 1:90 a ring can be obtained only with protein amounting to 300 mg. per hundred cubic centimeters. The globulin is obtained in a similar fashion by the use of ammonium sulphate. The albumin is computed by subtracting the amount of globulin from the total protein. The globulin divided by the albumin gives the so-called protein quotient.

MALAMUD, Iowa City.

EXTENSION OF EPIDEMIC ENCEPHALITIS TO THE SPINAL CORD. E. ILLING, Monatschr. f. Psychiat. u. Neurol. **82**:177 (March) 1932.

Illing reports the case of a woman, aged 40, who showed signs of a parkinsonian syndrome and muscular wasting resembling that seen in the Duchenne-Aran type of muscular atrophy. The patient had had an acute attack of epidemic encephalitis ten years previously, with signs of a parkinsonian state developing immediately afterward. The symptoms remained practically stationary for four years, following which they began to increase in severity. Three and a half years later the condition of the patient became much worse. At that time weakness of the muscles of the face and hands was first noted. These symptoms gradually became more pronounced during the next two and a half years, then seemed to come to a standstill. The left temporal muscle, the left masseter and the muscles of the ball of the left thumb showed the most marked atrophy. There were slight atrophy of the left half of the tongue, weakness of the left side of the palate and transient difficulty in swallowing. Fibrillary twitchings were occasionally observed in the affected regions. Illing points out that spinal involvement is by no means a rare sequela of epidemic encephalitis. However, he was able to find only one case, that of Reys, which presented changes similar to those observed in his case.

ROTHSCHILD, Foxborough, Mass.

THE BIOLOGIC DIAGNOSIS OF CYSTICERCUS. C. RIZZO, Riv. di pat. nerv. **41**:193 (Jan.-Feb.) 1933.

Rizzo describes the case of a man, aged 61, who for four months had had attacks of jacksonian epilepsy involving predominantly the facial muscles of the right side and, at times, the right upper extremity. As a result of the convulsive seizures the patient had dysarthria, which was transitory following the attacks, and slight but permanent weakness of the right extremities. The cerebrospinal fluid revealed lymphocytosis, which increased from 12½ to 102 cells per cubic millimeter. Eosinophilic cells had increased from 10 to 40 per cent in the last specimen. In this, as in three previously reported cases diagnosed during life and confirmed by autopsy, the diagnosis of Cysticercus was made exclusively on the basis of eosinophilia in the cerebrospinal fluid.

In describing the changes in the cerebrospinal fluid caused by Cysticercus cellulosae in the brain, Rizzo discusses the differential diagnosis from Echinococcus infestation of the brain. He stresses the importance of eosinophilia of the cerebrospinal fluid and insists that this may suffice for the diagnosis of Cysticercus. He describes a technic for detecting the pleocytosis of the cerebrospinal fluid and particularly its eosinophilic component.

FERRARO, New York.

EXTRAPYRAMIDAL DISTURBANCES IN OLIVOPONTOCEREBELLAR ATROPHY. HANS JOACHIM SCHEERER, Ztschr. f. d. ges. Neurol. u. Psychiat. **145**:406 (April) 1933.

There has been emphasis recently by the French, notably Guillain, on the part played by the cerebellum in extrapyramidal diseases. The occurrence of hypertonicity in these states is explained by an inhibition of the dentatorubral system through removal of the cerebellar cortex. Scheerer studied four cases of olivopontocerebellar atrophy to test this hypothesis. In all he found changes in the substantia nigra and basal ganglia. These were very severe in two cases, which showed symptoms referable to the basal ganglia. In the other two cases, which showed severe cerebellar changes and fewer changes in the substantia nigra and basal ganglia, the extrapyramidal symptoms were not evident. For this reason it was thought that the cerebellum does not play a very important rôle in the production of extrapyramidal disorders. So striking were the changes in the substantia nigra that Scheerer claims this to be a constant part of the picture of olivopontocerebellar atrophy. The changes in the striatum are similar to those of Huntington's chorea. The changes in the substantia nigra are looked on as a "premature local senility."

ALPERS, Philadelphia.

STATISTICAL AND ETIOLOGIC STUDY OF ANOSMIA. F. J. COLLET, Rev. d'oto-neuro-opht. **11**:1 (Jan.) 1933.

Ninety-five clinical cases in patients varying in age from 15 to 72 years were the basis of this study. The conditions were classified as traumatic anosmias, anosmias of nasal origin and anosmias in patients with pathologic conditions of the nerves. Eighty-two cases were in the first group, the larger number of which presented atrophic rhinitis, polyps and hypertrophic rhinitis. In the traumatic group, the anosmia occurred especially often from falls on the neck or occiput and the forehead, and was brought about by tearing of the olfactory fibers or by pressure from effusion of blood. The third group embraced cases of tabes, hemiplegia and neurosis. Two anatomic conditions dominate the pathology of the olfactory nerve: the tenuousness of the fibers penetrating the cribriform plate and the location of the cellular bodies of the olfactory neurons in the nasal mucosa itself. The first explains the vulnerability of the olfactory apparatus not only to trauma but also to nervous diseases that originate in a meningitic process. The second explains the persistence of anosmias of nasal origin from a transitory acute process. The olfactory organ is poorly protected against external noxious influences because it is in direct contact with the external air. The mucosa contains the olfactory cells; their destruction is irreparable and results in definitive anosmia.

DENNIS, Colorado Springs, Colo.

THE CEREBROSPINAL FLUID SUGAR. G. STRAUBE, Monatschr. f. Psychiat. u. Neurol. **82**:305 (April) 1932.

According to Straube, the normal sugar content of the cerebrospinal fluid varies from 45 to 65 mg. per hundred cubic centimeters. These figures were arrived at by studying eighty-five patients suffering from conditions such as schizophrenia, manic-depressive psychosis, functional reactions, psychopathy and idiocy. Sixteen patients with dementia paralytica and a smaller number of patients with other organic diseases of the nervous system were also investigated. In dementia paralytica the figures ranged from 42 to 67 mg. per hundred cubic centimeters. The lowest values were obtained shortly after treatment with malaria. Most of the results were within normal limits in tabes dorsalis, epilepsy, multiple sclerosis, syringomyelia, tumor of the brain and postencephalitic conditions. Three patients with symptomatic psychoses showed an increased amount of sugar in the cerebrospinal fluid. Low figures were obtained in cases of meningitis, but the results bore no relation to the number of cells or to the other changes in the cerebrospinal

fluid. In a group of nine patients with senile psychoses, seven showed high values for sugar. The author thinks that this increase was perhaps caused by disturbances of the blood-cerebrospinal fluid barrier.

ROTHSCHILD, Foxborough, Mass.

MORPHOGENESIS OF THE BRAIN. C. JUDSON HERRICK, J. Morphol. **54**:233 (March 5) 1933.

The fundamental structure of the brain stem is uniform throughout the vertebrate series because of the constancy of an apparatus of adjustment to the environment which is common to vertebrates, namely, peripheral connections and their internal relationships. Other parts of the brain are more variable because more elaborate mechanisms of correlation and integration are necessary as the behavior pattern in higher forms becomes more complicated. The chief apparatus of correlation and integration is elaborated in the brain stem between the olfactory bulb and other primary sensory centers. Here the tissue is plastic, not dominated by any single sensorimotor system, and is the meeting place of descending and ascending sensory pathways. From this point the complex thalamic and cortical adjustors emerge. The pallium is not a primary constituent of the vertebrate brain, but gradually emerged from the more ancient subpallial type of organization. Several stages in the progressive differentiation of the cortical structure and connections may be seen in the amphibian forebrain, but the process is not consummated. Amphibia have no cerebral cortex, though cortical primordia are evident here and in various fishes. Static concepts of architypes are replaced by a more dynamic treatment of morphogenesis.

WYMAN, Boston.

VASOMOTOR SYNDROME OF THE LABYRINTH. H. PROBY, Rev. d'oto-neuro-opht. **11**:182 (March) 1933.

Many vestibular disturbances that are not associated with definite pathologic changes are often overlooked. Whatever their cause, all may be traced to a circulatory disturbance. The reflex may be direct (foreign body or cicatrix) or may arise from a chronic infection, suppurating focus or hepatic insufficiency. Often no cause is found. An illustrative case is that of a married woman, aged 41, who suddenly experienced a sharp attack of vertigo and vomiting, which confined her to bed for eight days. The attack was ameliorated by the application of leeches to the mastoid region, but the vertigo, though less intense, persisted. Three years previous to the attack the patient suffered loss of consciousness for a few seconds. In other respects the history and all examinations gave completely negative results, except in the internal ear. Bone conduction and the reaction to cold irrigations of the ear were much reduced. Under treatment designed to modify the labyrinthine circulation, the vertigo diminished, and the normal excitability of the labyrinth returned. It is the evolution of the disease that differentiates the cases of circulatory disturbances from those of chronic infection of the labyrinth. The lesions disappear in the former and persist in the latter.

DENNIS, Colorado Springs, Colo.

THE TIME OF APPEARANCE OF EPILEPTIC SEIZURES IN RELATION TO AGE, DURATION AND TYPE OF THE SYNDROME. HELEN HOPKINS, J. Nerv. & Ment. Dis. **77**:153 (Feb.) 1933.

The periodicity of epileptic attacks has been observed by Gowers, Langdon-Down and Brain, who found that from 3 to 4 a. m. is the period of greatest frequency and that 7 a. m. and 8 p. m. are also time peaks that are unaltered by the administration of drugs. The study by Patry of 31 adults with 1,013 attacks confirmed these findings. Hopkins investigated 302 cases of the epileptic syndrome; 209 patients were over and 93 under 15 years of age; 155 cases were classified as idiopathic, and 149 as organic. Like preceding investigators, she found that the period of sleep and the period of waking from sleep represented the time of greatest

incidence. Seizures tend to occur within narrower limits of time in younger patients. No significant difference was observed between the idiopathic and organic type. The author draws attention to experimental evidence of increased brain volume and increased intracranial pressure during sleep, together with the observation of Sager and others that the disappearance time of a physiologic solution of sodium chloride is decreased during sleep, indicating an increase in the avidity of the tissues for water at that time.

HART, Greenwich, Conn.

INCOMPLETE FACIAL HEMIATROPHY OF SYMPATHETIC ORIGIN. G. WORMS, Rev. d'oto-neuro-opht. **11**:99 (Feb.) 1933.

Following the healing of an extensive mastoid wound, a young man began to experience paresthesia of the face on the side of the wound and, within three months, facial hemiatrophy was noticed. The atrophy was limited to the soft tissues and was accompanied by localized sweating of the forehead, cheek and parotid region, hyperesthesia of the two upper branches of the fifth nerve and increase of the nasofacial reflex. Increase of retinal arterial tension, absence of the oculocardiac reflex, enophthalmia and two bald spots on the temporal region on the same side were observed. Worms believes that these troubles arose from a disturbance of the vegetative system, caused by irritation from the mastoid scar. In support of the assumption that the sympathetic system was involved, another case is reported which was not due to trauma but followed repeated attacks of frank sympathalgia. In neither case was it a typical trophoneurosis, involving both the soft and the skeletal parts. Cases of facial atrophy of traumatic origin have been reported by Weekers, Leriche and others. In certain cases removal of the superior cervical sympathetic ganglion has been followed by good results.

DENNIS, Colorado Springs, Colo.

FURTHER INVESTIGATIONS ON THE DEVELOPMENT OF THE HORTEGA MICROGLIA. K. VON SANTHA and A. JUBA, Arch. f. Psychiat. **98**:598 (Jan.) 1933.

Continuing previous work (abstracted in ARCH. NEUROL. & PSYCHIAT. **29**:902 [April] 1933) von Santha and Juba studied embryo rats weighing 200, 170, 140, 120, 100, 75, 50, 35, 20, 15 and 5 mg. They investigated: (1) the stage at which the microglia first appear, (2) the genesis of the microglia and (3) their relationship to the appearance of gitter cells. The first appearance of microglia was observed at the 15 mg. stage; that is, between the eighth and ninth day. It coincided with the first appearance of vascularization in the central nervous system. Gitter cells were not observed until the 120 to 140 mg. stage; this is regarded as indicating that the gitter cells are not stages in the development of mature microglia. The authors believe that these cells develop from the microglia for the purpose of taking care of the catabolic products during development. The fact that the earliest microglia appear at the same time as the first stages of vascularization points to a mesenchymal origin of these cells. This is also supported by the fact that the first microglia are observed in close contact with the first vessels.

MALAMUD, Iowa City.

RESPIRATORY FAILURE AND THE DRINKER RESPIRATOR IN POLIOMYELITIS. EMIL SMITH, J. A. M. A. **100**:1666 (May 27) 1933.

The author believes that in any case of poliomyelitis with or without a rise in temperature respiratory muscle paralysis may develop within twenty-four hours. The most important indication for placing a patient in the respirator is a diminished vital capacity, which can easily be determined by the counting test, and as soon as this indication becomes apparent there is nothing to be gained by waiting for a fully established picture of respiratory muscle paralysis. Ten centimeters of negative water pressure in the respirator is sufficient to start with in cases of children under 10 years of age, and the respiratory rate of the machine should

correspond to the normal respiratory rate of the patient. The effect of the respirator in cases of respiratory muscle paralysis is an immediate and miraculous improvement, but prognosis should be withheld for at least five days in spite of the apparent improvement. The ability to breathe freely and without effort is not an indication for removing a patient from the respirator. The only indication is the ability to cough. Patients removed from the respirator without being able to cough may later contract atelectasis with bronchopneumonia.

EDITOR'S ABSTRACT.

OBSERVATIONS ON INDIVIDUAL SENSITIVENESS TO PAIN WITH ESPECIAL REFERENCE TO ABDOMINAL DISORDERS. EMANUEL LIBMAN, J. A. M. A. **102**:335 (Feb. 3) 1934.

Libman discusses individual sensitiveness to pain, substitution symptoms and radiations of pain. Some other important subjects, such as sensitiveness of diseased blood vessels and pains due to this, can only be touched on. Besides summarizing his earlier publications, the author gives a survey of recent investigations. Theoretical questions are not discussed. Many methods of gaging sensitiveness to pain have been suggested. The simple test that he employs is carried out by first pressing the thumb against the tip of the mastoid bone and then slipping the finger forward and pushing against the styloid process. Pressure on the normal mastoid bone causes no pain and therefore serves as a control. It is important not to rub the bone, because rubbing the periosteum of any bone is apt to evoke pain. Pressure in the direction of the styloid process is painful to some persons and not to others. The sensitive point is really not the styloid process but a branch of the auricularis magnus nerve. Algesimeters do not appear to be of service in practical clinical tests.

EDITOR'S ABSTRACT.

THE PHYSIOLOGICAL AND CLINICAL RESULTS OF DIVISION OF THE VAS DEFERENS. A. CLIFFORD MORSON, Brit. M. J. **1**:54 (Jan. 14) 1933.

Morson finds that there is no justification for the old belief that division of the vas deferens has effect on either the size or the function of the prostate gland. The quantity of the secretion is unaltered, and the man who has had both vasa divided is unaware of any change in the act of coitus. The vesiculae seminales gradually shrink and atrophy. As far as can be determined, there is no change in the shape or size of the testes. What happens to the spermatozoa that continue to be produced is problematic. The final phase, in all probability, is the impotence of the testis to produce any sperm cells at all. The technic of operation is given and can be carried out by any medical practitioner with a knowledge of minor surgery, but it is essential that it be performed efficiently, otherwise serious complications may result. It has been fairly well proved that infection from the bladder neck and prostatic urethra reaches the testis by way of either the lumen of the vas or lymphatics within its walls. Thus this operation becomes of importance in checking the spread of infection to the testes from the bladder and urethra.

FERGUSON, Niagara Falls, N. Y.

CLINICAL ASPECTS OF ABDOMINAL PAIN. F. M. POTTENGER, J. A. M. A. **102**:341 (Feb. 3) 1934.

Pottenger states that exaggeration of a sensory stimulus, whether it is transmitted centrad over cranial or spinal afferent nerves from the skeletal structures or over the efferent systems that accompany the sympathetic and parasympathetic nerves to the internal viscera, may lead to discomfort or pain. Pain is the automatic danger signal that tells the patient to beware, for the normal mechanism is irritated in an abnormal manner, or even its integrity may be threatened. In considering pain, it is necessary for the clinician to remember that the perception of painful stimuli is bound up with the patient's physiologic stability and that the complaint

of sensory changes will differ greatly in different persons. What may be a painful stimulus to one person may not be to another. The author discusses the following phases of pain as they pertain to abdominal pain: the visceral nervous system, the nature of visceral pain, the segmental nature of visceral pain, the difficulty of comprehending reflex effects, the difference in acute and chronic pain and the usual location of pain for the principal abdominal viscera

EDITOR'S ABSTRACT.

BLOOD SUGAR MOBILIZATION—EFFECT OF EPHEDRINE IN CHRONIC ENCEPHALITIS. ISIDORE FINKELMAN, J. Nerv. & Ment. Dis. **77**:345 (April) 1933.

Finkelman reports the investigation of the glycemic and circulatory responses to ephedrine in 14 cases of chronic encephalitis, and 8 controls consisting of 2 manic-depressive patients, 1 nonpsychotic normal person and 5 mentally defective patients. Ephedrine hydrochloride was used as a sympathicomimetic drug to observe its effect on blood sugar mobilization in chronic encephalitis. Seven of the 14 blood sugar curves obtained in chronic encephalitis showed a reversal of the ephedrine action. The failure of mobilization of blood sugar after stimulation with ephedrine may be due to lesions involving, to some extent, the central sympathetic nuclei. The blood sugar curve was raised after injection of ephedrine in patients treated with parasympathetic depressants, such as atropine and scopolamine. This confirms the existence of parasympathetic predominance in chronic encephalitis. The author also concludes that it contraindicates the presence of local changes in the liver in this disease.

HART, Greenwich.

THE INFLUENCE OF SECTION OF THE VESTIBULAR NERVE ON THE SINGLE-SHOCK REFLEX FLEXOR RESPONSE. E. G. T. LIDDELL, K. MATTHES, E. OLDBERG and T. C. RUCH, Brain **55**:232 (June) 1932.

In experiments in which the eighth nerve was sectioned in cats, less increase in excitability of the ipsolateral than of the contralateral flexor was found. Support is given to this view, as there may be a decrease in inhibition, such as occurs when the spinal cord or certain tracts within it are severed. Section of the vestibular nerve on one side changes the pattern of reflex response from that which obtains in the customary "intercollicular" preparation; thus, the conclusion of Camis that labyrinthectomy alters the normal activity of, e. g., the flexor and extensor muscles of the leg is supported. The spontaneous rolling movements of the animals immediately after operation may be due to the disturbance of a preexisting balance of inhibition due to the influence of the uninjured labyrinthine mechanism of the other side.

MICHAELS, Boston.

BILATERAL SUPRARENAL HEMORRHAGE AS A CAUSE OF SUDDEN DEATH IN EPILEPSY. LEGRAN A. DAMON, Psychiatric Quart. **6**:438 (July) 1932.

Only twenty cases of bilateral suprarenal hemorrhage have been reported, and of these a definite history of epilepsy has been found in three. This percentage suggests a relationship, although whether the convulsive phenomenon tends to produce the bleeding, or whether the same factor which causes the paroxysms also causes the hemorrhage, cannot be determined. The case reported is that of a man at a colony for epileptic patients who suddenly complained of cramps and diarrhea. The next morning he collapsed and died within a few hours. Autopsy showed extensive hemorrhages in both suprarenal bodies; the kidneys were not implicated. The normal cellular relationships were completely destroyed and replaced by extravasated erythrocytes. Although both medulla and cortex were involved, the latter was less completely destroyed by the hemorrhagic process.

DAVIDSON, Newark, N. J.

REFLEX RELEASE OF FLEXOR MUSCLES BY SPINAL SECTION. E. G. T. LIDDELL, K. MATTHES, E. OLDBERG and T. C. RUCH, Brain **45**:239 (June) 1932.

Since transection of the spinal cord of a decerebrate preparation enhances the flexor reflex, the present study was performed to discover the spinal tracts the section of which is responsible for the release of the flexor center. Enhancement of the flexor reflex was obtained only in lesions of the cord on the same side in experiments on twenty cats. After complementary postmortem anatomic studies, it was found in the posterolateral region that the lateral reticulospinal tract is the path by which impulses descend in the decerebrate preparation to check flexor activity. In the sections through the anterior region of the cord, the vestibulospinal and the ventral and medial reticulospinal tracts are concerned in the release of the flexor centers.

MICHAELS, Boston.

PATHOLOGIC CHANGES IN THE EAR IN NIEMANN-PICK'S DISEASE. JOSEPH G. DRUSS, Arch. Otolaryng. **15**:592 (April) 1932.

For the first time in the literature Druss reports the pathologic changes in the ear in a case of Niemann-Pick's disease. He found the typical foam cells in the submucosa of the tympanum and antrum, in the bone marrow and perivascular spaces of the temporal bone, about the nerve trunks in the anterior auditory meatus and in the purulent exudate of the ear. The typical pathologic observation of foam cells throughout the body was made at autopsy. The similarity of this disease to Gaucher's disease and Brunner's case of Gaucher's disease affecting the temporal bone is noted. Both of these diseases are due to abnormal lipoid storage and both follow disease of the reticulo-endothelial system.

HUNTER, Philadelphia.

CEREBRAL PENETRATION OF BISMUTH: EXPERIMENTAL RESULTS WITH IODOBISMUTOL. AND SOME OTHER BISMUTH PRODUCTS. P. J. HANZLIK and JEAN B. SPAULDING, Am. J. Syph. **16**:335 (July) 1932.

As the result of a series of experiments on animals, Hanzlik and Spaulding conclude that products containing electronegative bismuth are more efficient and more penetrating in the treatment for cerebral syphilis than preparations containing electropositive bismuth. They found, for example, that proprietary bismuth-iodide preparations (the metallic radical of which is an anion) appeared in the brain in the quantity of 0.05 mg. per hundred grams of tissue, whereas the commonly used sodium bismuth tartrate, which contains electropositive bismuth, appeared in only one-half that concentration.

DAVIDSON, Newark, N. J.

IDENTIFICATION OF LUCAS'S ALPHA EXCITABILITY. W. A. H. RUSHTON, J. Physiol. **75**:445 (Aug. 10) 1932.

In this paper reasons are given for concluding that the alpha excitability in muscle is the excitability of the normal muscle fibers themselves. The isometric twitch is practically identical whether elicited by an alpha or by a gamma stimulus. The rapidity of the alpha twitch excludes the possibility of identifying it with Bremer's neuromuscular contracture, which is very slow. By several different methods the alpha excitation is shown to arise at the cathode, owing to the closing (not opening) of a current. The alpha contraction is not a local Tiegel's contracture, but is propagated. A hypothesis is put forward by the author to explain qualitatively the dependence of the excitation time on the nature of the electrodes and on the relative independence of nerve.

ALPERS, Philadelphia.

NEUROTROPISM OF SPIROCHAETA PALLIDA. GEORGE RAIZISS and MARIE SEVERAC, Arch. Dermat. & Syph. **26**:271 (Aug.) 1932.

Emulsions of the organs of syphilitic mice were injected into the testes of normal rabbits. Record was kept of the organ which supplied the emulsion in each case. It was noted that the extract of mouse brain was able to penetrate into the brain of the rabbit, producing cerebral syphilis in the latter animal, whereas the ordinary strain of spirochete taken from organs other than the brain did not have this property. Furthermore, the ability to penetrate the brain of the rabbit seems to be conserved by the strain of spirochetes taken from the emulsion of the brains of syphilitic mice, even though transplanted through a chancre. This offers a method for the chemotherapeutic investigation of drugs intended to attack and destroy the spirochete after it has penetrated into the central nervous system of the rabbit.

DAVIDSON, Newark, N. J.

NEUROVASCULAR CARDIAC DISORDERS: POSSIBILITIES IN THE FIELD OF MANAGEMENT. FREDERIC J. FARNELL, J. Nerv. & Ment. Dis. **75**:217 (March) 1932.

The author employed the circular compression wave apparatus of Rosett in the treatment of fourteen patients suffering from various degrees of arterial hypertension and cardiovascular disorders, and of some patients with anxiety states and other neurotic phenomena. He concludes from the gradual lowering of the pulse rate and blood pressure in these cases, that the apparatus applied periodically to the body, producing a sleep different from that induced by drugs or hypnotic suggestion, seems to have a physiologic effect on the cardiovascular system. This effect shows itself in the relaxation of both voluntary and involuntary muscles and in a diminished state of conductivity of the entire nervous system.

HART, Greenwich, Conn.

EMOTIONAL DISTURBANCES AMONG CHILDREN. ELLEN B. SULLIVAN, J. Juvenile Research **16**:56 (Jan.) 1932.

Since emotional disturbances in children are often the result of complex environmental factors—social, educational or physical—it is often necessary (and often forgotten) to modify these external forces before trying to change the child. Sometimes doing nothing is the best form of treatment, especially when any sort of action focuses the child's attention on a disgrace or humiliation which he ought to forget. Often the adult companions, especially parents and teachers, will repeatedly emphasize these errors and perpetuate the attitude of inferiority. Manifestly these adult points of view must be changed before any progress can be made.

DAVIDSON, Newark, N. J.

POLYGLOBULIA WITH OR WITHOUT ERYTHROSIS IN CERTAIN SYNDROMES OF THE HYPOPHYSIS AND TUBER CINEREUM. G. GUILLAIN, P. LECHELLE and R. GARCIN, Ann. de méd. **31**:100, 1932.

Three cases of dystrophia adiposogenitalis and one case of acromegaly are reported in which the number of red blood cells was higher than 6,000,000 per cubic millimeter. In the last case the blood picture returned to normal after the removal of a hypophyseal adenoma. Based on personal observations and other reports of polycythemia (type Geisböck), combined with erythremia, obesitas, polyuria and disturbances of growth, the authors advance the hypothesis of the existence of a center for the regulation of the hematopoietic equilibrium in the subthalamic region.

WEIL, Chicago.

FAMILIAL BILATERAL ACOUSTIC TUMORS. LOUIS MINSKI, J. Neurol. & Psychopath. **12**:289 (April) 1932.

Three members of the same generation of a family are reported. One had double acoustic neuroma with several similar smaller growths on the seventh and twelfth cranial nerves. Another died with tumors on the roots of the spinal cord, which were examined only macroscopically. The third was diagnosed by neurologic examination, roentgenologic evidence of internal auditory meatal involvement, and histologic examination of one of the neurofibromas removed from the arm. In the two cases of the acoustic condition increased protein was noted in the cerebrospinal fluid with zone I curves. All three patients gave a history of a blow preceding the onset of symptoms to which no significance was attached. The remainder of the family gave no suggestive signs or history of neurofibromatosis.

SPERLING, Philadelphia.

CONVULSIONS IN PELLAGRA. H. SEELERT, Monatschr. f. Psychiat. u. Neurol. **82**:337 (May) 1932.

The author observed unusual convulsive seizures in three patients with schizophrenia who acquired pellagra. The attacks took the form of continuous clonic contractions of the facial muscles, with a mixture of choreiform, myoclonus-like and clonic movements of the trunk and extremities. One patient also had a generalized tonic convulsion. In the first case the seizures began after symptoms typical of pellagra had been noted. In the other cases the convulsive phenomena occurred early; their close resemblance to the attacks already observed was an important factor in making the diagnosis, for these patients failed to present characteristic cutaneous changes or pronounced intestinal disturbances. A fatal outcome occurred in each case. Histologic examination disclosed changes similar to those found in cases of pellagra by recent investigators.

ROTHSCHILD, Foxborough, Mass.

REMOVAL OF RIGHT CEREBRAL HEMISPHERE FOR INFILTRATING GLIOMA. W. J. GARDNER, J. A. M. A. **101**:823 (Sept. 9) 1933.

The author states that, of three patients whose right cerebral hemispheres he removed, the last two died within thirty-six hours of hyperthermia, and the first is alive and well twenty-one months after operation. From these results it appears that this operation entails a grave risk, but that if the tumor is completely removed, an indefinite continuance of life is possible. As the speech areas of the brain are in the opposite hemisphere in right-handed persons, no demonstrable speech or intellectual impairment follows the operation. The preservation of function may be quite remarkable, as is shown by the patient whose case the author reports. The earliest motion to appear after operation was the automatic movement of walking. This indicates that the function present in the left leg is due to basal ganglion innervation and is not due to impulses from the homolateral hemisphere traversing the direct pyramidal tract.

EDITOR'S ABSTRACT.

SPONTANEOUS SUBARACHNOID HEMORRHAGE. H. F. DOWLING, Am. J. M. Sc. **185**:469 (April) 1933.

Three groups of spontaneous subarachnoid hemorrhages are presented as an etiologic classification: (1) blood dyscrasias; (2) extrinsic diseases affecting the blood vessels of the subarachnoid space, and (3) intrinsic abnormalities of the blood vessels. In the series of twelve cases reported, 33 per cent were associated with syphilis, and 42 per cent with a blood pressure above normal. The most prominent symptoms were headache and unconsciousness. Among the signs, hyperactive deep reflexes, choked disks and unequal reflexes were the most prevalent. It is suggested that in every case of coma it is important to suspect subarachnoid

hemorrhage and, therefore, to perform a lumbar puncture. The best treatment is to apply measures that will relieve intracranial pressure. The twelve cases that form the basis of the study are summarized individually.

MICHAELS, Boston.

A CASE OF OSTEITIS DEFORMANS TREATED WITH PARATHORMONE. G. H. COLT and A. LYALL, Brit. M. J. **2**:10 (July 1) 1933.

The authors report the treatment in a case of osteitis deformans by the administration of from 5 to 10 units daily of parathyroid extract (Collip), with periods of intermission, during two years. The treatment was augmented by sodium biphosphate, 30 grains (1.95 Gm.), taken three times daily, well diluted in water, to depress the absorption of calcium in the intestine, to decrease the calcium content of the serum and to promote the excretion of calcium in the urine. Progress was noted by a series of roentgen films, which suggest definitely that there is now less calcification in the skull than there was two years before when treatment began. The patient feels better and has been able to carry on her housework and go out walking without fatigue, which had not been the case before. Her general appearance also is markedly better.

FERGUSON, Niagara Falls, N. Y.

VASCULAR CRISES. DAVID RIESMAN, Am. J. M. Sc. **185**:29 (Jan.) 1933.

A broad, comprehensive view of vascular crises is interestingly presented in this article, with stress on fundamental concepts. Of the general vascular crises, sudden rises and falls of blood pressure are most important. In the majority of instances the local vascular crises are of spastic type and result from a constriction of the vessels. Those of the brain and eye are of special significance to the neurologist. Little is known of vascular spasm in the lung; the author holds to the angiospastic theory of angina pectoris. The vascular crises in the lower extremities, depending on constriction or vasodilatation, comprise intermittent claudication, Raynaud's disease, scleroderma and erythromelalgia. Further interpretations, in addition to Sir Thomas Lewis' views, are necessary to explain the cause of pain in angiospasm. Increasing stress and strain of life is a most important factor in thrombosis and embolism.

MICHAELS, Boston.

PATHOLOGIC ANATOMY OF MYASTHENIA GRAVIS. V. TRONCONI, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **35**:93, 1933.

Two cases of myasthenia gravis were subjected to histologic investigation. The central and vegetative nervous systems were studied, with special attention to the extrapyramidal system. Tronconi found changes in the nerve cells and glia in the nucleus of the hypoglossal nerve in one case. The changes were minimal. Pathologic changes were found in both cases in the vegetative nuclei, diencephalon, rhombencephalon, striatum, dentate nucleus, red nucleus and substantia nigra. The changes, in the author's opinion, were insignificant and cannot be regarded as the anatomic substratum of the disease. He cites Berlucchi, Emma and himself as having recently reported similar changes in the structures mentioned in the cases of persons who died from diseases other than those of the nervous system.

KESCHNER, New York.

ALZHEIMER'S DISEASE WITH PICK'S ATROPHY IN THE PARIETO-OCCIPITAL LOBE. M. LIEBERS, Arch. f. Psychiat. **100**:101 (June) 1933.

Liebers describes a case of a presenile psychosis with autopsy. The clinical picture was typically that of Alzheimer's disease. The gross pathologic condition, however, was like Pick's atrophy of the parieto-occipital lobe. Microscopic examination showed, distributed throughout the cortex, numerous senile plaques and

Alzheimer fibrillary changes. There were no swellings of the cells and no argentophil globules. The cortical architecture was well preserved. Clinically there were none of the outstanding features of Pick's disease. In discussing the case the author emphasizes the point that although the atrophy in Alzheimer's disease may sometimes simulate grossly the appearance of Pick's atrophy, nevertheless both clinically and histologically the two diseases can be differentiated.

MALAMUD, Iowa City.

SACROCOCCYGEAL CHORDOMAS IN CHILDREN. ALBERT H. MONTGOMERY and IRVING J. WOLMAN, Am. J. Dis. Child. **46**:1263 (Dec.) 1933.

The authors review the literature in 108 cases of chordoma, 53 of which were situated in the region of the sacrum and coccyx. In their own cases, of which 3 are reported, the ages were 3½ months, 22 months and 3 years, respectively. Two patients died. The third was apparently in a satisfactory condition at the time of discharge. Early surgical intervention is considered to be the most satisfactory form of treatment. In most cases the tumors recur. Irradiation may be of some value, particularly in the relief from pain. The diagnosis can be made finally only by finding characteristic cells in the fluid obtained by exploratory puncture. The prognosis in these cases is considered to be bad because of the malignancy of the tumor.

WAGGONER, Ann Arbor, Mich.

FIELDS OF VISION IN CONNECTION WITH INTRACRANIAL LESIONS. ADAMS A. MCCONNELL, Brit. M. J. **2**:226 (Aug. 5) 1933.

The importance of exact localization in operations on the brain is stressed. In neurologic diagnosis, a visual defect must be looked for as carefully as one looks for rigidity and tenderness in the right iliac fossa. The anatomy of the visual paths is discussed concisely. The visual pathway may be divided into five sections: (1) the intracranial part of the optic nerves, (2) the optic chiasm, (3) the optic tract, (4) the visual radiation and (5) the striate area of the occipital lobe. This paper deals with tumors affecting the visual pathway, involving the optic tract and the optic radiation, particularly in relation to the temporal lobe of the brain. Seven cases are reported, showing the visual field and the site of the lesion as determined at operation.

FERGUSON, Niagara Falls, N. Y.

THERAPY FOR PNEUMOCEPHALUS INTRACRANIALIS TRAUMATICUS. L. GUTTMANN, Ztschr. f. d. ges. Neurol. u. Psychiat. **140**:92 (June) 1932.

Treatment for traumatic pneumocephalus has usually been conservative, with no operative intervention if possible. Operative procedures have been carried out in some cases, with varying results, for the purpose of repairing the defect between the sinus and the brain. Guttmann reports a case in which there was a large cystic collection of air in the left frontoparietal region. With conservative treatment, the spinal fluid ceased to escape in four months. Later, epileptic attacks developed, and it was because of these attacks that Foerster dissected out the cyst and the adhesions. The attacks ceased after operation. The appearance of epileptic attacks, therefore, may be a good indication for operative intervention in this disorder.

ALPERS, Philadelphia.

EFFECTS OF EXTIRPATION OF THE CAROTID "CORPUSCLE" ON THE RETINAL CIRCULATION. VAN HOONACKER, Ann. d'ocul. **170**:89 (Jan.) 1933.

After extirpation of the carotid "corpuscle" there is an increase in the systolic and diastolic blood pressure of the retina, not only on the side operated on, but also on the opposite side. If the carotid "corpuscle" of the other side is removed later, the retinal blood pressure increases on both sides. The resulting effect on the retinal circulation is permanent. The increase varies in different subjects;

in all cases, systolic pressure generally increases slightly more than diastolic pressure, the increase averaging from 10 to 15 for the diastolic pressure and about 20 for the systolic pressure. If both "corpuscles" are extirpated, these figures may be approximately doubled. When a change in ocular tension occurs it is likely to be a slight bilateral hypertension. BERENS, New York.

THE REPAIR OF SKULL DEFECTS BY A NEW PEDICLE BONE-GRAFT OPERATION. R. WATSON JONES, Brit. M. J. **1**:780 (May 6) 1933.

A new operation is described for the repair of defects in the vault of the skull by a pedicled bone graft. The graft of bone is obtained from the outer table of the skull, keeping the plane of division strictly to the diploe and leaving the inner table intact and uninjured. The advantages of the operation are that the graft may be cut any size, it has the same curved contour as the skull, it lives through its pedicle, there is excellent fixation of the graft to its bed, and no pressure is exerted on the meninges and cerebrum. A case is reported in which the operation has been successfully performed.

FERGUSON, Niagara Falls, N. Y.

OPTIC ATROPHY AND RETROBULBAR NEURITIS IN THE COURSE OF EPIDEMIC ENCEPHALITIS. E. LAZARESCO, Ann. d'ocul. **170**:348 (April) 1933.

Lazaresco examined a patient who, in the course of a febrile condition associated with herpes labialis and polyneuritis, diagnosed as epidemic encephalitis, lost vision in the right eye. At this date, examination of the fundus gave negative results. Forty days later the disk was atrophic. Eight months later the right optic nerve was atrophic; there was no perception of light in this eye, and there was accentuated rigidity.

In a patient with spastic paraplegia following epidemic encephalitis, a unilateral central scotoma developed (ophthalmoscopic examination gave negative results), followed by transitory paralysis of the extrinsic and intrinsic muscles of both eyes. BERENS, New York.

NONTRAUMATIC HEMATOMA OF THE DURA MATER. J. A. BARRÉ and JEAN MASSON, Encéphale **28**:81 (Feb.) 1933.

Clinical and pathologic studies lead to the grouping of the following symptoms and signs as characterizing nontraumatic hematoma of the dura: bilateral frontal headache without predominance of side, in spite of unilaterality of the bloody extravasation; photophobia; torpor of variable intensity; vertigo and vestibular reactions; true reflexes of defense (little spontaneous movements, abrupt or light and of variable amplitude and almost incessant); absence of convulsions and of true paralysis; light contracture of musculature but without a true Babinski sign or at least until the terminal phase; incontinence of sphincters, even during waking hours; fleeting rigidities of the neck with an inconstant Kernig sign; variable pupillary status; absence of papillary stasis. ANDERSON, Los Angeles.

EXAMINATION FOR DIPLOPIA WITH A RED GLASS. A. DE JAEGER, Ann. d'ocul. **170**:339 (April) 1933.

The frequency of the occurrence of diplopia in the commonly employed methods of examination has been studied by de Jaeger. Complete diplopia, similar to that found when paralysis of a muscle is present, is discovered in some patients who are not troubled by diplopia or vertigo. Almost all of this type of diplopia is noted in the lateral fields, rarely in the vertical field; the images are always homonymous, appearing on each side, at equal distances from each other. De Jaeger believes it probable that this diplopia is due to unequal movements of association (one eye being in abduction, the other in adduction), which is more marked for the adducted eye. BERENS, New York.

CHORDOTOMY FOR SARCOMA OF THE LEFT BUTTOCK AND PERINEUM. J. MONTENEGRO and J. O. MATOS, Rev. Assoc. paulista de med. **1**:213 (March) 1932.

Montenegro and Matos report an advanced case of sarcoma of the left buttock and perineum, in which a right-sided chordotomy (Spiller-Martin-Frazier operation) was done. Pain was completely relieved, and the patient was made absolutely comfortable. The day after the operation, there were retention of urine and paralysis of the right leg and left arm. Twenty days later, however, the retention of urine, as well as the paralysis of arm and leg, had completely disappeared. There remained only a slight motor weakness of the right leg.

EDITOR'S ABSTRACT.

DOUBLE ACOUSTIC NEURITIS AND INCOMPLETE CEREBELLAR PYRAMIDAL SYNDROME FOLLOWING MUMPS. J. HELSMOORTEL JR., ALBERT MINNE and L. VAN BOGAERT, J. de neurol. et de psychiat. **32**:720 (Oct.) 1932.

The authors present a syndrome of right hemiparesis with increase of the tendon reflexes and a positive Babinski sign on the right, a left cerebellar condition and bilateral loss of hearing without vestibular disturbances following mumps. The spinal fluid was under increased pressure. There were 14 cells, 44 mg. of albumin, and 56 mg. of dextrose. The Pandy test was positive; the Wassermann test was negative, and the colloidal benzoin test showed a meningeal type of reaction. Presumably the symptoms were consequent on meningitis.

WAGGONER, Ann Arbor, Mich.

SOME EFFECTS OF TRAUMA ON THE NERVOUS SYSTEM. C. M. H. HOWELL, Lancet **1**:115 (Jan. 16) 1932.

The importance of trauma in precipitating latent organic neurologic conditions is emphasized by Howell by the citation of several cases. One of his patients had been apparently in perfect health until thrown from a horse. He was placed in bed, where he remained for two days. On getting out of bed he found difficulty in walking. Examination at this time revealed well developed tabes dorsalis. The author also cites a case of severe injury to the body, the head being unaffected, followed by the sudden appearance of an unmistakable attack of dementia paralytica.

DAVIDSON, Newark, N. J.

ASEPTIC OTOGENOUS SEROUS MENINGITIS: CURE. LACROIX, Rev. d'oto-neuro-opht. **10**:742 (Dec.) 1932.

The case reported is that of a young girl in whom, in the course of chronic otitis, meningitis developed. The cerebrospinal fluid contained 150 cells and 70 cg. of albumin. No organisms were found on culture. Immediate radical mastoidectomy with wide exposure of the dura was done, and a large cholesteatoma and a perisinous abscess were revealed. A fixation abscess was produced, and rapid recovery followed. Lacroix emphasizes that the operative results are better when the fluid is clear and the cellular elements are intact, and that prompt and thorough operation is important.

DENNIS, Colorado Springs, Colo.

SOLAR RADIATION IN RELATION TO ENDEMIC GOITER. JAMES H. SMITH, Arch. Int. Med. **50**:76 (July) 1932.

The distribution of endemic goiter in the United States, New Zealand and India is studied and compared with the intensity of solar radiation in these countries. The similarity between the areas is striking, and the relationship is further confirmed by a determination of the iodine content in the water in these zones. Smith concludes that there tends to result from a deficiency of solar radiation a deficiency of iodine in the thyroid gland as the result of defective irradiation of the air, soil, water, food or skin.

DAVIDSON, Newark, N. J.

Ovarian Follicle Hormone Therapy in Ovarian Insufficiency and the Menopause. Bruce Webster, Am. J. M. Sc. **184**:822 (Dec.) 1932.

In a series of twenty cases, consisting of ten cases of menopause (two artificial and eight natural) and of ten cases of amenorrhea (five of insufficiency of ovarian follicles and five of the anterior pituitary ovarian type) treated with ovarian follicle hormone (Allen-Doisy units) intravaginally, it was thought that there was a definite improvement in the clinical symptoms except in the last type. Evidence as to the physiologic activity of the hormone was shown by the reestablishment of uterine bleeding.

Michaels, Boston.

Conditioned Reflexes: Physiologic Considerations. Paul Meignant, Encéphale **27**:857 (Dec.) 1932.

This experimental and theoretical discussion is based on the fundamental work of the Pavlov school. Meignant believes that the concepts of excitation and inhibition, with their corollaries, are inadequate to explain all observations. The concept of induction, whereby a reflex activity governs in part the reflexes of antagonistic groups, is developed. Also, there is much discussion of the rôle of the cortex in the analysis of reflexes.

Anderson, Los Angeles.

Neuroblastoma of the Adrenal with Multiple Metastases. Jacob Klein, Am. J. M. Sc. **184**:491 (Oct.) 1932.

In view of the rarity of this disorder, a case in a girl, aged 2½ years, is described. Seven weeks prior to admission to the hospital, there were serious symptoms, and although there were no characteristic findings, a malignant growth was suspected. The pathologic diagnosis was primary sympathicoblastoma of the right suprarenal medulla with metastases to the lymph nodes, calvarium, left humerus, ribs, orbital fat tissue, liver, lungs and spleen.

Michaels, Boston.

Action of Epinephrine on the Arteries of the Brain and Lung. Hsi Chun Chang, Arch. f. d. ges. Physiol. **231**:200, 1932.

Isolated arteries of the brain and of the lung were perfused, and the resistance of the arteries was measured. Epinephrine had only a slight effect on the arteries of the brain; in 150 experiments, 65 showed contraction of the arteries; 18, dilatation and 67, no change. The same artery contracted in one experiment and dilated in another; these differences of reaction could not be explained. The arteries of the lung usually dilated under the influence of epinephrine.

Spiegel, Philadelphia.

Educational Achievement of Delinquent Boys. Vera Chase, J. Juvenile Research **16**:189 (July) 1932.

Chase studied the scores of 165 boys at a California school for delinquent children, using the new Stanford achievement tests. She found that in all subjects these children were inferior to public school children, but that their highest ratings were in reading and geography. In the latter subject their progress in school from grade to grade approximated normal, probably because it was a source of interest to them. The lowest scores were made in arithmetic.

Davidson, Newark, N. J.

Abscess of the Frontal Lobe Due to Acute Frontal Sinusitis. T. Flournoy Wickliffe, Arch. Otolaryng. **15**:290 (Feb.) 1932.

This article is a short report of a case in which an external frontal operation was performed. Partial healing of the wound was followed by a frontal abscess and erosion of the posterior plate of the frontal bone; two months after the

operation this erosion caused a bulging of the frontal area. Incision was made, and the frontal abscess evacuated. Death followed. The author had advised earlier operation, which was refused.

HUNTER, Philadelphia.

THE INFLUENCE OF THE SYMPATHETIC ON THE CEREBROSPINAL NERVOUS SYSTEM. H. ALTENBURGER and D. M. RIOCH, Arch. f. d. ges. Physiol. **229**:473, 1932.

Stimulation of the sympathetic chain usually increases the chronaxia of the motor as well as of the sensory nerves. This effect is observed only in intact animals (cats). In decerebrated cats, stimulation of the sympathetic had just the opposite effect: the chronaxia was shortened. The mechanism of these effects of the sympathetic on the cerebrospinal nervous system is still uncertain.

SPIEGEL, Philadelphia.

AN UNUSUAL CASE OF MENINGITIS ASSOCIATED WITH HAEMATURIA. L. MARGARET MASTERMAN, Brit. M. J. **2**:834 (Nov. 5) 1932.

The points of interest in this case are that the initial symptoms resembled those of an acute hemorrhagic nephritis. The onset of meningeal symptoms was delayed. Clinical and laboratory evidence suggested a meningococcic infection, but this diagnosis was not proved. Rapid improvement followed encephalography. This was considered to be due to the breaking down of adhesions or to the releasing of an encysted area.

FERGUSON, Niagara Falls, N. Y.

ELECTROCARDIOGRAMS OF MENTAL PATIENTS. LOUIS LEVIN, J. M. Soc. New Jersey **29**:140 (Feb.) 1932.

Organic cardiac disease is found in 4 per cent of all persons in the general population. In the Trenton State Hospital, organic cardiac disease was diagnosed in 14 per cent of the patients. In almost a third of these, a diagnosis was made solely on the basis of electrocardiographic findings. Levin believes that more attention should be paid to the physical study of persons who are mentally ill.

DAVIDSON, Newark, N. J.

OLIGOSYMPTOMATIC RUDIMENTARY TABES. R. H. RAMIREZ, Arq. brasil. de neuriat. e psiquiat. **15**:61 (Feb.-March) 1932.

Ramirez sums up his view in the words of Austregesilo: Two or three important symptoms of the tabetic series persuade us to establish a diagnosis of incipient tabes; if these symptoms are of long standing and of slow evolution, the condition is abortive or oligosymptomatic rudimentary tabes, which is the diagnosis that should be made.

EDITOR'S ABSTRACT.

THE AMEBOID CELLS, THEIR TIME OF APPEARANCE. A. HOLZER, Ztschr. f. d. ges. Neurol. u. Psychiat. **140**:272 (June) 1932.

Holzer found ameboid cells in the cortex in a case of trauma of the head in which the patient died six hours after the accident. Necropsy was performed two hours after death. The ameboid cells were present often around the punctate hemorrhages in the brain, indicating that they were formed before death. Holzer cites this case to prove that ameboid cells are not always a postmortem change.

ALPERS, Philadelphia.

DIAGNOSTIC ERRORS OF NEUROSURGERY. A. MONTEIRO, Arq. brasil. de neuriat. e psiquiat. **15**:72 (Feb.-March) 1932.

Monteiro cites several diagnostic errors of neurosurgery and emphasizes that many diagnostic errors could be avoided if the general practitioner would, in the

early course of a disorder, avail himself of the special services of the roentgenologist and the ophthalmologist, for conditions in Brazil are not essentially different from those in European countries.

EDITOR'S ABSTRACT.

CORNEAL LESIONS AND THE POSTERIOR CERVICAL SYNDROME. H. VIALLEFONT, Ann. d'ocul. **170**:893 (Oct.) 1933.

Viallefont reports three cases of recurrent ulcerative keratitis with hypesthesia of the cornea. The syndrome of the posterior cervical sympathetic nerve caused by a cervical arthritis was also present in these cases. In cases of hypesthetic keratitis of unknown origin, Viallefont believes that the syndrome of the posterior cervical sympathetic nerve should be considered.

BERENS, New York.

THE KETOGENIC DIET IN EPILEPSY. S. M. ALLAN, J. Ment. Sc. **79**:677, 1933.

A ketogenic diet was administered to twenty adults with epilepsy for sixteen months. In thirteen cases there was a reduction in the number of fits; in two the number was unchanged, and in five there was an increase. Seven patients improved mentally; the other thirteen showed no change. The author states that although his results are not so favorable as those obtained by Helmholtz they suggest that the method is worth trying.

KASANIN, Howard, R. I.

A STUDY OF THE LAURENCE-BIEDL SYNDROME. G. WEILL and PAYEUR, Ann. d'ocul. **171**:173 (Feb.) 1934.

Weill and Payeur report a case of the Laurence-Biedl syndrome in an infant with polydactylism, hyperfunction of the anterior lobe of the hypophysis, heredosyphilitic chorioretinitis and a salt and pepper appearance of the fundus. The Bordet-Wassermann reaction was positive. The authors believe that the retinitis pigmentosa in this case is without doubt of heredosyphilitic origin.

BERENS, New York.

TREATMENT OF MALIGNANT DISEASE BY PITUITRIN AND THEELIN. W. STEWART, Brit. M. J. **1**:98 (July 16) 1932.

Stewart was unable to confirm the findings reported by Susman in October 1931 on the successful treatment of malignant disease by the administration of pituitary and ovarian hormone, with a low carbohydrate diet. In no patient treated by this method was any appreciable change observed.

FERGUSON, Niagara Falls, N. Y.

THE EFFECT OF THE GROWTH HORMONE ON THE BRAIN WEIGHT-BODY WEIGHT RATIO. H. S. RUBINSTEIN, Anat. Rec. **53**:265 (July 25) 1932.

On the basis of the experiments reported in this article, the brain is less responsive to the growth hormone than the body as a whole. It is therefore concluded that the brain weight-body weight ratio is decreased by the administration of the growth hormone.

COBB, Boston.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Feb. 15, 1934

A. W. STEARNS, M.D., *Presiding*

SOME NEW METHODS IN THE STUDY AND THE TREATMENT OF MIGRAINE. DR. WILLIAM G. LENNOX, Boston.

Riley has spoken of "neurologia irredenta," i. e., neurologic diseases which in a large part have been lost to the other specialties of medicine. Migraine may be called a medical "no man's land," possession of which depends on investigation and research. Three papers that have appeared in the last year point the way to three aspects of the problem which especially deserve exploration. Draper reported significant observations on the emotional and psychologic states in a group of patients with migraine. Riley, Brickner and Kurzrok secured new evidence concerning the rôle of the secretions of the pituitary gland in the production of headaches. Dickerson relieved seven patients with periodic hemicrania by section of the middle meningeal artery and the accompanying nerve.

Members of the Neurological Unit at the Boston City Hospital, with the aid of the Macy Foundation, are conducting research into the causes of headaches. With the collaboration of Dr. von Storch and Dr. Pool, observations are being made on the spinal fluid and blood pressure during headache and on the period of relief which comes with administration of ergotamine tartrate. With Dr. and Mrs. Gibbs, we are studying the cerebral blood flow during and after headache, Using a thermo-electric blood flow recorder inserted in an internal jugular vein. We also measure the gases of the blood in this vein. Dr. Solomon is studying the sympathetic nervous system, with special reference to the electrical resistance of the skin.

The most complete report possible at this time concerns our experience with the use of ergotamine tartrate in aborting attacks of migraine. This drug has been used in thirty-five cases. Prompt cessation of the headache occurred in all but four. Some of the patients received the drug only once, but eight have taken it for prolonged periods, up to two years. It is most effective when given intravenously, though hypodermic administration usually gives good results. A detailed report appears in the *New England Journal of Medicine* (**210**:1061, 1934). Ergotamine tartrate promises relief for many patients who are not helped by the ordinarily used sedative drugs. Furthermore, it affords a mean for the study of migraine, because its effect seems to be specific for headache of this type.

DISCUSSION

DR. C. A. MCDONALD: For years, ergot and ergotine have been used for the prevention and cure of migraine. Dr. Courtney at the Massachusetts General Hospital, fifteen years ago, said that migraine was due to vasomotor disturbance; he used ergot, but although enthusiastic at first he finally agreed that phenobarbital gave more relief. Is not this new drug a derivative of ergot? As much as 20 or 30 minims (1.2 or 1.9) of ergot every two hours has been used in the treatment for migraine in the past years without promising results.

DR. D. GREGG: Dextrose given intravenously has been known to give prompt relief from headache. What is the explanation?

DR. T. J. PUTNAM: Little is known of the anatomy of the blood supply in the head and the physiology of pain in the head. They are difficult to investigate.

As Dr. Lennox pointed out, it is hard to induce patients with headaches to come to a hospital, probably because the symptoms are seldom entirely incapacitating and the patients have a dread of being made worse. The functional element is of striking importance. Investigation of the physiology of headaches in animals is, of course, extremely difficult. There is no way of measuring. It is hard to determine whether they have a headache, but not impossible. However, the anatomy and physiology are beginning to fit together better. The upper spinal nerves and the trigeminal nerve have many fibers the origin and termination of which are difficult to make out. It is fortunate that Dr. Lennox has decided to investigate this matter. The systematic investigation by Dr. Lennox is an admirable work, and the cooperation of all neurologists can make this investigation a success.

DR. A. W. STEARNS: Have the social histories been investigated in these cases?

Dr. W. G. LENNOX: Ergotamine tartrate is a specific alkaloid of ergot. It is standardized and stable, and the results of its use are therefore more constant than those secured with the fluidextract. The observation of the beneficial effect of intravenous injections of dextrose solution is important if confirmed. The rationale for this treatment is not clear. Many patients find that the ingestion of sugar seems to precipitate headaches. The reduction of intracranial pressure should not be helpful, for during headache the cerebrospinal fluid pressure is normal or low. The social and emotional histories are indeed important. For example, the woman whose headaches date from her marriage was wedded at an early age to a man not her equal in order to escape from her mother.

THE MECHANISMS OF RECOVERY IN A PATIENT WITH PARANOIA. DR. MORRIS YORSHIS.

History.—A man, aged 29, who had been married for five years, but had no children, a Catholic, a photographer, the third of four siblings—a sister being two years younger—was admitted to the Worcester State Hospital a little over three years ago. His complaints were: (1) For six months the family had whispered about him behind his back; (2) he suffered from headaches and insomnia; (3) the family forced him to eat when he refused his meals; (4) they were antagonistic and conspired against him in other ways; (5) he had lost some of his proofs by confiding in a physician.

The patient felt that he would become insane because of: (1) the stock market crash, (2) clients' suicides and (3) the effect of a visit to an insane cousin. He became increasingly quarrelsome, irritable, sullen and suspicious. He was afraid of an Italian client, who had lost money in the stock market. He worried about impotence and accused his wife of infidelity. He had found tablets of corrosive mercuric chloride in a medicine cabinet where his phenobarbital was. "Coffee had the same taste as the mercury tablet." Six weeks before commitment he had injured his scrotum while playing football, and a brother fed him poison to affect his sexual ability permanently. He had considered killing the brother for interfering with his happiness. He described a system of attempts on the part of his people to kill him. The sensorium was intact; the intelligence quotient was 118. Insight was poor.

History.—The family history was moderately positive for alcoholism, migraine and insanity in the immediate family. The father was irritable, impatient and impulsive, and an inveterate alcoholic addict, especially when affected by migraine. The oldest brother, an alcoholic addict, died in the thirties following a debauch. A sister, two years younger than the patient, had been operated on for tumor of the brain, but none was found; her migraine had been less severe since operation.

The patient's past history is presented in the accompanying table.

Physical Examination.—The patient's height was 5 feet and 7 inches (170.18 cm.), and his weight, 114 pounds (51.7 Kg.). There was a postoperative scar on the left side of the neck; the reflexes were hyperactive.

Past History in Chronologic Order

Year	Events in Personal History	Related Items	Age	Illnesses
1901	Instrumental delivery; prolonged labor; mother wished for a girl		Birth	
1902	Weaned very early (6 mos.); fussy; hated bottle; constipated	Because of illness mother favored him	1	Mumps
1903		Sister born	2	Tuberculous gland removed
1904	Suspicious of oldest brother whom mother held as an example (9 yrs. older than patient); strong dislike for father		3	
1905			4	Measles
1906	Troublesome, fussy, nervous, fidgety and restless; little devil; courageous; always in mischief; tied up in yard by mother	Sister would be given freedom of yard and brother allowed to go anywhere	5	
1907	Started to school but left because he missed his sister; obstinate; temper tantrums; full of self-pity		6	
1908	Started to school again with sister; no chums	Brother-sister relationship very tense	7	
1909	Always in scraps; no sense of humor		8	Chickenpox
1910	Would tag after brother (4 yrs. older) but then shifted to sister		9	Diphtheria
1911	Nose picking stopped; enuresis began		10	
1912			11	
1913			12	
1914			13	
1915			14	
1916			15	
1917	At high school; work above average; popular in school; member of student council; objected to confession; cynical		16	
1918	Proctor of schoolroom; leader in physical training		17	
1919	Just got by in school; discontinued church		18	
1920	Extremely mortified if in a group of men who were talking of their various sex experiences	When brother spoke to patient about masturbation, patient asked him to drop subject	19	
1921	Graduated from high school		20	
1922	Intercourse with girl chum of sister; relations unsatisfactory, having orgasm too soon or not at all; found he couldn't satisfy girl when she was aroused; worked for father; left home because of quarrel with father but came back later	Marriage, he believed would rid him of the nasty habit of having relations with men	21	
1923			22	
1924	Patient and brother courted same girl; she preferred patient; sent away on trip; patient upset in accident; driver killed	Settlement arranged by brother	23	Laceration of scalp; skull fracture
1925	Married—knew wife two months; took correspondence course on "How to Get Along with People"; would get irritable and fire his best men; became a stock salesman	This would cause difficulty between him and father and brother; oldest brother died. (He was very successful in the stock market)	24	
1926	Patient was managing business of father; was also a stock salesman	Brother sick; nervous breakdown; stayed home for two years; depressed and lost interest in things about him	25	
1927	Patient kept wandering, doing business in various cities; kept moving from place to place because of restlessness and difficulty with men		26	
1928	As soon as brother returned, patient thought he was being eased out; hired men would report to brother and not to the patient	Oldest brother bequeathed $9,000 to mother; both father and brother lost it in the business	27	
1929	Losses in stock market heavy; jealous of brother who became head of photography business	Clients lost money and concern the patient worked for failed; his best friend committed suicide	28	

The following sex activities were noted: 1903-1906, sucking of thumb and picking nose; 1911, beginning of enuresis; 1916-1918, mutual masturbation; 1920, nail biting; 1922, first heterosexual experience; 1925, attempt at heterosexual adjustment.

Clinical Course.—Direct active therapy was begun in February 1931.

Feb. 3, 1931: "Father used to beat me mercilessly, and I felt like killing him. It was a big family; everybody seemed to make a success and then they would go to pieces. I went to pieces before I ever succeeded."

February 4: "This candy my wife brought me wasn't poisoned. The seal wasn't broken. I know all the time we were together she put up with an awful lot. She is upset in her sexual relations. She has had it for a number of years and would want it badly. She and my brother are together an awful lot."

March 18: Escaped.

April 14: Returned from escape.

June 14: On vacation with wife. Behavior during first week good. Second week paced the floor, was sullen and suspicious and felt that he was being watched.

July 18: "If I could go out and be left alone, I would be O.K. It gets on my nerves to be watched. They keep you posted."

July 20: "I must have an unusual mind. Keep anybody in here for a length of time and they are apt to lose their mind. My father drank heavily. I always fought with him. He just spoiled everything."

July 21: "I don't know why I should be in here. Either one of them is worse than me. My father or brother. I made a mess of things by blaming my wife."

July 27: "I am still worried about this damn sex business. I don't always want to be this way. At the time I got medicine in New York. It seemed to help but now I see it didn't."

August 14: Intercourse with wife successful.

August 21: Headache less severe and less frequent. Brother no longer visited patient.

August 23: Dream. "I was being attacked by a snake. The snake was long and thin. Just as I was about to shoot it with my gun, the snake injected some poison into my left ankle but before I was ruined or dead, I shot the snake." Explained: "The snake in the dream was my brother. He is a snake in the grass. Even in the dream he has the upper hand."

August 26: "It always struck me funny how my brother could read my mind. What did my sister mean by saying, 'Hurry up and get out of here?' Maybe it would be better if I let her have the children. She'd be better off if she did get rid of me on account of this sexual business."

Indirect passive therapy was begun in September.

September 4: Dream. "I was riding on a Pullman. I occupied the lower berth and my brother the upper. I thought I would never get to my destination. The wheels kept grinding—grinding." This reminded him of the many trips he and his brother took together. "When I was down in Mobile one of the salesmen masturbated me."

September 7: "Please don't tell my wife about these sex experiences with men. My brother seemed to know I was masturbating. When I was 13 or 14 another boy masturbated me. At 18 a salesman working for my father roomed with me, and we masturbated each other for about a month."

September 28: Dream. "I was having intercourse with myself. My penis was a yard long, and it was very round and was in my mouth. This disgusted me so much that I had an emission."

October: Headaches increased in frequency. Wished no longer to have treatment such as he had the previous month. Asked to have treatment again.

November: Brother left home.

December: Insurance settlement.

Jan. 17, 1932: Released from hospital for an indefinite visit.

The direct active approach to the patient's problem was an important factor in his recovery.

Comment.—The hereditary and familial character of migraine is exemplified here, the father and three of the four siblings being affected. It is of clinical interest that the attacks increased in frequency and severity whenever the patient

discussed painful topics or when the interview was postponed. The evidence for organic injury to the brain following the automobile accident, in which the patient sustained a lacerated scalp and questionable fractured skull, is not adequate. Changes in personality, irritability and headaches are common sequelae, but a study of the personality before the injury was ample proof that these difficulties had existed from early youth.

In all his contacts with men the patient was on the defensive. He did not trust the men whom he employed and could not get along with those with whom he worked in the stock broker's office. His hatred for his father and brother made it difficult for him to make friends with men, especially those in authority. The strong attachment to the mother and sister is obvious.

The "best friend," who committed suicide, may have been responsible for the latent paranoid dynamism becoming active in the patient. He considered himself a murderer, but this was traceable to the time of the automobile accident which resulted in the death of the driver, as well as the death wishes aimed at the father and brother.

The sexual difficulties were most conspicuous. Whenever he spoke about homosexuality he was very much abashed, suspicious and reticent, which showed where the real trouble lay. The patient's marriage was both an attempt to put a barrier between him and his homosexuality and possibly a means of homosexual gratification, but he found his tendencies breaking through again in the "psychosis" (the return of the repressed in paranoid form—Freud). The behavior during the time that homosexuality was discussed showed conclusively that he preferred to return to his wife rather than face the underlying difficulty. The sister attachment is an example of how the regard for the mother was transferred to the sister. The sexual relations were with the sister's chum (a symbolic substitute).

His loss of money in the stock market was an upsetting circumstance. The patient always looked on money as power; he believed that he needed this to get the proper recognition from his mother. The timely occurrence of some of the attacks of migraine helped the patient to recognize their significance. The impotence disappeared without the aid of drugs.

The brother's departure from the home and the economic security gained by the insurance settlement were important factors in the patient's recovery, but the flight from homosexuality probably was the real reason for his adjustment.

DISCUSSION

DR. C. M. CAMPBELL: It is extremely interesting to follow such a case as that of Dr. Yorshis; the collection of such cases may enable one to see what the factors are which modify the course of the disorder, which cause stabilization at an inferior level or which favor improvement. In the present case there must be considered the problem of the sex life, the difficulty of later adult adjustment, the question of a constitutional anomaly and the patient's early relations with the parents, especially the early relations with the father and the reaction to being beaten by the father. The relation to the brother involves a great complexity of factors. When the psychosis broke forth he threw responsibility on the outer world. One hopeful sign was that in the beginning he was able to establish a good rapport with the physician; he was not shut off in his own reveries; he was willing to discuss the whole affair. Then he came to a readjustment. The great difficulty in learning the history of a paranoid condition is that it takes such a long time to make the necessary review. A diagnosis of "paranoid" tends to condemn a patient. To size up all the assets of a case like this and to estimate the liabilities is extraordinarily difficult. It is good to have a concrete example to think over. Continuous discussion may help the patient. Often patients with a distorted attitude derive great benefit from the cooperation of the physician, and without such intensive study the delusions would be more crippling than they need be.

DR. WILLIAM HEALY: The story of this man with his homosexual experiences and ideas leads me to say that I am more and more impressed with the relationship of homosexuality, in its various phases, to the development of paranoid trends.

The patient tries to repress the trend, feels guilty about it and projects his own feelings, and minor or major ideas of reference and delusions readily develop. I have seen many illustrations of this, and as one I would cite the case of a young man who was brought to me by a business man who said that the officials over the boy were concerned about him because he was too good. He worked overtime to be sure that everything he did was right. The patient came from a good family and was ambitious. He had had homosexual experiences with a man of considerable social standing, and unfortunately he found these experiences not unpleasant, although they conflicted so much with his conscience. He tried to make up for his feelings of guilt by being overly good, at the same time worrying about his own temptations and becoming almost ascetic in the endeavor to repress them. Soon he was wondering what people suspected about him. An effort was made to get him to a psychiatrist, but instead the family sent him traveling to get him away from the homosexual approaches of the man in his social set. It was learned before long that he had a definite mental upset and later was sent to an institution with a diagnosis of paranoid dementia praecox.

Of course, by no means all who have homosexual ideas or experiences show such mental abnormalities. There may be various causes for the development of the abnormalities, but it seems as if that regulator of ideas and conduct, the conscience, has something to do with it. The distinction that one sees between boys, coming perhaps from private schools where homosexual practices have been in vogue, according to whether or not they have highly developed feelings of guilt is really extraordinary. One of the main tasks in preventive psychiatry is relief from guilt feeling. About persons who take up homosexual life and follow it without any compunctions, I need say nothing. It is obvious that they float along without any mental upsets. They have no tremendous sense of guilt and no deep repression.

DR. M. R. KAUFMAN: It is interesting that one who is not a psychoanalyst should observe a case of this sort and get so much material that demonstrates the relationship of homosexuality to paranoid symptoms. Freud first made the point that paranoid delusions were related to homosexuality, particularly in his brilliant analysis of the Schreber case.

DR. EARL ZINN: This case, so ably presented by Dr. Yorshis, shows again the influence of the homosexual factor in paranoia. There is only one point which I wish to emphasize, and it relates to the mechanism of recovery. Seven months after therapy began the patient presented the first clearly homosexual dream. Following this there was a surge of homosexual material. So impressive was this that it led Dr. Yorshis to change his technic and, I think, thereby prevented a further development of the paranoid condition. Fortunately, at approximately this same time two events related to the reality situation occurred. The patient's brother, to whom there was a strong homosexual attachment, left home, thus removing what heretofore had been a constant source of unconscious temptation; a settlement was made with the insurance company which solved the patient's financial problem, at least temporarily, and also eliminated a secondary motive for retaining his illness. Confronted in the therapeutic situation with the prospect of facing his homosexual wishes, but with flight to his home equally dangerous so long as the brother remained in the situation, his dilemma remained. The alteration in the reality situation offered a way out, and he took it. What the future holds in store for him depends, I think, on whether or not he is again faced with a situation in which homosexuality is reactivated to the point where it seems likely to overcome the repressing forces.

DR. D. GREGG: I hope that Dr. Yorshis will be able to report on this case sometime in the future. For recovery from a paranoid condition, thirteen months is a short period. Dr. Yorshis speaks of the patient as having been weaned at an early age, at 6 months. I doubt if pediatricians would consider 6 months an early age for weaning. The history is consistent with recovery from a depression with paranoid symptoms. I should like to know later if recovery has been maintained.

DR. NEILS ANTHONISEN: This case is of interest as it seems to take an intermediate position between the transitory paranoid conditions that seldom come under the observation of psychiatrists but run their course relatively unnoticed and the types that become chronic without having had periods of full remission. The improvement of the patient at the time Dr. Yorshis discontinued his intensive study suggests that he acted wisely. The consequence was, of course, that he had to sacrifice a deeper insight into the dynamic associative relations of the case. One aspect, however, of considerable significance, viz., the temperament of the patient, lent itself to a more thorough investigation than I think Dr. Yorshis has given it. It seems to me that the history gives indication of a noteworthy difference in this respect between the present case and those of the chronic type of paranoid condition. In the latter one finds the paranoid affect, to express it simply, more or less constantly sustained, with little tendency to relaxation, while in Dr. Yorshis' case there was a definite undulation. There was an increase and a decrease, the latter tending toward a relief of tension until a new rise was precipitated. Was there an increase of symptoms with the discussion of the patient's sexual life? If I am not wrong, the history did not indicate that there was. One would certainly have expected an increase, and the lack of it must be considered an unusual feature.

DR. M. YORSHIS: Dr. Campbell asked about the patient's attitude toward his father. It was mainly one of contempt. One does see paranoid conditions not related to homosexuality, although many are, as Dr. Healey said. The time relation of the discontinuing of discussion of homosexuality to the money settlement was not important, as the patient was unaware of it; and even if the brother had not left home the patient would have been sent out of the hospital. I cannot answer the question that Dr. Gregg raised. As soon as the patient was weaned, he had gastro-intestinal upsets. Finally, regarding diagnosis, the question is whether this was a case of depression with paranoid trends or one of a paranoid condition. All the physicians who saw the patient thought that he had paranoia. There was a decided increase in paranoid symptoms as soon as homosexuality was discussed.

SCHIZOPHRENIA IN ONE OF IDENTICAL TWINS. DR. J. KASANIN, Clinical Director, State Hospital for Mental Diseases, Howard, R. I.

Work in the field of mental disease is complicated by the fact that there are at least two sets of variable factors, the original endowment and the environmental factors. In identical twins there is an opportunity to study the specific influence of environmental factors because the original endowment, with the innate tendencies and dispositions, is constant. Parker believed that the occurrence of schizophrenia in identical twins points to a germinal origin of the disease, but conservatively remarked that perhaps it is accidental that there is no report in the literature of a case in which a schizophrenic breakdown took place in only one of identical twins. The reason why such cases have not been reported is, in my opinion, that the cases in which schizophrenia took place in both twins were more dramatic and attracted more attention. I shall report a case in which only one of identical twins was affected by the disease and the other has remained well for eight years.

A. B., a white man, aged 29, a cabinet maker, single, was admitted to the State Hospital for Mental Diseases, Howard, R. I., on Aug. 25, 1931, and was discharged on Nov. 14, 1933. The diagnosis was dementia praecox, paranoid type.

The patient was transferred from the psychopathic division of the Providence City Hospital because for several years he had had ideas of persecution and had made threats to kill. He believed that the government was shadowing him, and was therefore unable to keep a job for the five years prior to hospitalization. He was born on Nov. 10, 1901, in Providence, R. I. Birth was normal and at full term. The early development was normal. Walking, talking and dentition occurred at the usual ages. There was nothing significant about the patient's

childhood. The boy started to attend school at 6 and completed grammar school at 14. He graduated from a technical high school at 18. He was an excellent student and wished to go to college, but the family could not afford it. The patient had pneumonia at 6. Both twins had measles at 5 and mumps at 6. At 16, both twins began to experience a hearing defect, especially in the left ear. Their health was otherwise good. After leaving school the patient obtained work as a machinist in a large industrial concern. He worked there for two years and was laid off during the depression. After this he began to do odd jobs as a cabinet maker and carpenter. He always managed to earn between $25 and $35 a week. A few years before his admission to the hospital a piece of steel became embedded in his left eye. A cataract formed over the traumatic area so that vision was impaired.

The patient was described as idealistic, interested more in writing than in manual work. During the senior year in high school he showed extreme interest in religion. He was considered seclusive and reticent, and had few friends. He spent all of his leisure time with the twin brother, to whom he was strongly attached. After graduation from high school, the interests of the two brothers became divergent. The patient became more and more absorbed in religion, whereas the twin was more interested in worldly matters. For two years the patient studied the Bible. He went to church frequently, until at the age of 20 he began to feel that religion did not satisfy his needs. The patient never went out with girls, and the family ascribed this to natural reticence and bashfulness. When compelled to go into society, strangely enough, he was active and was "the life of the party." At the age of 20 he shifted his interest to literature; he studied Shakespeare and decided that he should write. All his leisure time was spent in this manner. His essays were heavy and morbid; publishers rejected them. In an attempt to write something more acceptable, the patient decided to write sketches for vaudeville. These, however, were far too serious. Instead of confessing inability to write, the patient began to feel that some person or organization was preventing him from making good. He thought that people were trying to blackmail him and prevent the publishers from accepting his manuscripts. Three years before admission he complained of being shadowed by detectives. As he walked he looked around suspiciously and acted so strangely that he was picked up several times by the police. In September 1930, he decided to go to Boston where he would have more peace and would not be disturbed by detectives who prevented him from achieving success. His money ran out during the winter, and he had to go to work as a dishwasher to support himself. If any one tried to approach him, he decided that this must be a detective. In the early spring of 1931 he started for New York, but on the way stopped in Hartford and obtained a job as a dishwasher in a restaurant. He was laid off in a few days and blamed the detectives for it. After he arrived in New York he obtained a few odd jobs, but most of the time he was without work and was hungry. He began to feel that everybody in New York was a detective. He let his hair grow long and looked like a backwoodsman. One day his sister, who is a manager in a department store in New York, saw him on the street and when she recognized him became much upset. She sent him back to Providence, and as soon as he arrived there he went to the police to complain about detectives. The police advised the family to have him committed to an institution.

On admission to the City Hospital he was sullen, suspicious and resentful, and told a story about being shadowed by many detectives. He was preoccupied, introverted, seclusive and extremely suspicious. He told the physician that five years previously detectives began to watch him and question him about his literary efforts. In a hotel where he lived, a large number of "snoopers" came in and filled the halls, corridors and streets and every nook and corner of Providence. They followed him so much and made his life so miserable that he had to go to another city. He went from city to city to escape their persecution. The patient stated that he must have been taken for somebody else and that up to the time he entered the hospital the detectives interfered with him, meddled in his business, questioned

him, etc. The work, he said, was done by government stool-pigeons, and the people who asked him for aid were also stool-pigeons. He was correctly oriented in all fields.

When transferred to the state hospital he repeated the same ideas of persecution. Most of the time he was extremely seclusive, evasive, suspicious and uncommunicative. Within a few months he began to be more communicative and took more interest in the environment. He spoke more freely about his delusions and mentioned many incidents to show how the various detectives tried to make his life miserable. He spoke of a young girl in the hotel who tried to "make" him and also "frame" him. He stated that she knew all his business, that she "tipped off" other people about him and sent detectives to arrest him in Boston when he happened to look in a window of a jewelry store.

In November 1931, the patient was allowed to go home for a few days. He was brought back several times as he had difficulty in adjusting himself in the community and in obtaining employment. On several occasions he annoyed various public officials, and it was necessary to send him back to the hospital.

C. D., the twin, had the same backgound as A. B. He also had impairment of hearing at the age of 16, and the condition was diagnosed as catarrhal deafness. In comparing the twins the family thought that C. D. was more active, vigorous and somewhat more extroverted than A. B. He seemed to be more alert and was able to maintain better contacts with people. Nevertheless, he was extremely attached to his twin brother, and the pair were inseparable. During his last two years in high school he was also extremely interested in religion. After graduation from high school an uncle in Detroit invited one of the twins to come there and work with him. A. B. did not express any enthusiasm about going to Detroit as he was too much interested in religion. For a long time the twins could not make up their minds to separate, but finally the economic depression forced a decision, and C. D. went to Detroit. Both brothers are good machinists and cabinet makers; C. D. rapidly became successful and within a few months was making as much as $100 a week as a tool maker. For two or three years he worked in Detroit, saved several thousand dollars and decided to complete his education. He went to a college of engineering in a midwestern city. On graduation he returned home, served a year of apprenticeship in a large electrical concern and then started a small business of his own. He is having a somewhat difficult time due to general business conditions but is energetic and hopeful and is trying to make the best of the situation. He is a keen, alert man, who looks exactly like the patient but is more aggressive.

A. B. weighs 161 pounds (73 Kg.), and C. D., 162 pounds (73.5 Kg.). Both have gray-blue eyes. The left eye of A. B. shows an opening of the iris in the 5 o'clock direction where the foreign body entered. There is slight opacity of the lens due to the foreign body and slight adhesion to the lens. Both brothers have impaired hearing, greater in the left ear than in the right.

There has been the usual striking similarity in the two brothers since early childhood. There have been the usual stories of their own parents not being able to tell them apart. At school it was deemed advisable to separate them and to place them in different classrooms. The family physician, Dr. A. C. Crocker, of Providence, believes that there was only one placenta when the twins were born, but he has no birth record. He has observed the twins throughout their lives and states that they have been strikingly similar since birth and that he could never tell them apart. The question which naturally arises at this point is whether or not they are identical twins. Most students of heredity agree that when twins are of the same sex and look strikingly alike the chances are that they are identical.

An analysis of the cases shows the great importance of environmental factors. One brother becomes more interested in religion than the other, although both went through a period of marked religious interest. C. D. went to Detroit and became a successful mechanic and later a professional man; "life smiles upon him," and he has made a satisfactory adjustment, overcoming the difficulties resulting from the present business depression. A. B. tried to write, did not succeed, was a failure

and projected his failure on the outside world, blaming the government and various people as being responsible for his failure. I think that this case illustrates unusually well the relative importance of environmental factors. The twin brothers have approximately the same endowment and their life careers were similar up to the age of 18, as there had been no variation in the environment. At the age of 18 the environment changed sharply. It became favorable for the one and just as unfavorable for the other. Although possessing the same endowment and the same early environmental forces, there have developed altogether different psychobiologic reactions. One has become a successful, well-adjusted man in his community. When the environment became bad for the other, who is presumably endowed exactly as his brother, he found a solution of his failure in a psychosis. Granting that there is some slight difference even in identical twins, a significant dynamic influence of environmental forces leading to the preservation of mental health in one twin and to the formation of a psychosis in the other seems to have been demonstrated.

DISCUSSION

DR. K. M. BOWMAN: Has the brother ever been examined by a psychiatrist? The fact that he is a successful osteopath does not mean that he may not have a psychosis or have had one in the past. It is also possible that one will develop later. I recall that at the time of Professor Parker's report I saw a young man whose condition I diagnosed as schizophrenia. He had an identical twin. I was able to see the twin and talked with him for an hour without eliciting anything. However, the social service department discovered that this brother had been a patient about eight years before at the Boston Psychopathic Hospital, and his condition had been diagnosed as schizophrenia. He had apparently made a fair degree of recovery, as did the patient mentioned.

It seems to me, therefore, that one needs to be very cautious in assuming that the case reported proves that one twin may have a psychosis and the other be normal. I also raise a question as to whether these twins are identical. The picture shown seems to indicate some differences in features and in finger prints. It seems possible that these brothers are not identical twins.

DR. D. GREGG: A number of years ago I had under observation a patient with neurasthenia who had a twin sister at a state hospital suffering from long-standing dementia praecox with deterioration. The similarity in appearance between these two patients was marked, and I consider that they were probably identical twins. On seeking information from the Boston Police Department, however, as regards identification through finger prints, I was told that if I submitted ten finger prints they could not pick out twins from the collection because there was so much difference between the finger prints of twins. I question whether one can possibly make a diagnosis of identical twins unless the history of a single placenta is obtained.

DR. J. KASANIN: Only with the history of one placenta can one prove identity. The brother was seen by me several times and appeared to be normal. However, he was suspicious. He knows about the usual occurrence of mental disease in both twins and is trying hard to fight the fatalism with which his own future is surrounded. One must not forget that he is under severe environmental stress, as everybody in the family expects him to break too; a slight deviation from the usual behavior would be immediately interpreted as a beginning mental disease. In spite of this careful observation by friends and family he is doing well, and I see no evidence of any mental disease in his case. Concerning the difficulty in recognizing identical twins, Dr. Holmes of the University of California writes that there is no way one can tell if twins are identical or not. The only time when finger prints are identical is when the eggs become separated very late; as a rule, identical twins do not have the same finger prints. One twin often does not marry. Twins are not really endowed in the same way. One usually is superior at the expense of the other.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Feb. 15, 1934

JAMES C. HASSALL, M.D., *Vice President, in the Chair*

TREATMENT OF PROGRESSIVE MUSCULAR DYSTROPHY WITH GLYCINE: DYNAMOMETRIC STUDIES. DR. BENJAMIN BOSHES and DR. ARTHUR WEIL.

We have studied six cases of progressive muscular dystrophy in children who were fed glycine. A dynamometer was used to record the changes in the strength of the muscles. Observations were made at intervals of from six to eight weeks for as long as twenty-two months. Two patients with far advanced cases showed no improvement that could be demonstrated clinically or dynamometrically. Three showed improvement, with increases of over 100 per cent in strength in some readings. Power increased generally when glycine was administered and decreased when this amino-acid was withdrawn. In the sixth case, while dynamometrically stronger, the patient was clinically weaker. Here the muscular dystrophy was complicated by Fröhlich's syndrome. The child gained weight more rapidly than he gained strength. He was obese, and the increase in power in the legs did not parallel the gain in the other muscles.

While the period of study is too short for definite conclusions we think that in not too advanced cases glycine offers a possible means of retarding or arresting the progress of the wasting in progressive muscular dystrophy.

DISCUSSION

DR. ARTHUR WEIL: Dr. Boshes carried out the major part of this investigation. If one compares our conclusions with those in the paper read by Dr. Reese and his co-workers before this society, one will notice that we are not as optimistic as they were, but, on the other hand, we could not be as pessimistic as others who have denied any good results from glycine. Our conclusions are about midway between those of the other authors. It seems that glycine is capable of restoring muscular strength to a certain level, but it may be noticed from our charts that following this increase of muscular power there was a rather constant level that did not show much variation. As Dr. Boshes pointed out, one cannot overlook such an improvement or claim that this was a natural effect of growth; one should ascribe it to the effect of the glycine. In drawing conclusions for the practitioner one should point out that no time should be wasted in far advanced cases of muscular dystrophies. The earlier the patient is treated the more favorable seem to be the results. Whether the disease can be cured we cannot say from an observation extending over only about eighteen months, but our work shows that at least the disease can be arrested during such a period.

DR. R. P. MACKAY: I wish to ask: 1. Were metabolic studies made on the excretion of creatine and creatinine? I am under the impression from Boothby's work that on the administration of glycine one notices at first an increased excretion of creatine, and as the medication is continued the excretion decreases. Is it possible that such a course of events could be correlated with an initial loss in strength followed by a subsequent rise in power as observed in these patients by Dr. Boshes?

2. Would a larger dose of glycine be more effective than the dose used in these cases? Some observers have used as high as 15 Gm. a day.

3. Would better results be secured by combining ephedrine or epinephrine with glycine? Such a combination seems to be beneficial in myasthenia gravis.

DR. SOLOMON B. MEYERSON, St. Charles, Ill.: Were metabolic or other studies made to determine the presence of any possible dysfunction of endocrine glands, such as the thyroid or the pituitary gland? Symptoms of Fröhlich's syndrome are mentioned in one case. Were roentgenograms made to determine the presence of any transformation of the sella turcica? Dr. Boshes stated that

in two cases epinephrine and pilocarpine were administered with no effect. What method was used to determine the effect of pilocarpine and epinephrine? Was the dynamometer employed, as in the cases in which glycine was administered? It was also reported that one patient was more alert at school following treatment with glycine. Were psychometric studies made in that case to determine if there was an actual improvement in the child's general intelligence and mental abilities?

I became interested in the myopathies in 1929 when at the Lincoln State School and Colony there came under my observation a mentally defective boy, aged 12, who presented a classic history and symptoms of infantile progressive muscular dystrophy. In addition, he had symptoms of adiposogenital dystrophy. Roentgenographic studies revealed an exceedingly small sella turcica and atrophic changes in the bones of the extremities. The sugar tolerance test gave normal results. Two years later (1931) there developed symptoms suggestive of hyperthyroidism, with a basal metabolic rate of + 31 and loss of about 30 pounds (13.6 Kg.) in weight, although the characteristic distribution of fat was still maintained. There were no instances of myopathy, abnormal adiposity or feeblemindedness in the family. The parents were divorced; the father was said to have been intoxicated at the time of the patient's conception. The patient was treated for several months with epinephrine and an anterior lobe pituitary preparation, without salutary effects.

DR. MEYER SOLOMON: Did Dr. Boshes consider the possibility that the psychologic effect on the patient during these studies might have stimulated him to greater effort than he would otherwise have made?

DR. BENJAMIN BOSHES: We made no metabolic studies in these cases. The patients belonged to another clinic and were all ambulatory, attending the Spaulding School for Crippled Children, and it was difficult to control them metabolically. Reports of metabolic studies are conflicting. As a rule, the creatine rises after the taking of glycine, and the creatinine may rise; after a given period the creatine begins to fall. How is this to be interpreted? Creatine is not a favorable element in the urine of these patients, and whether its increase should be taken as an index of improvement it is difficult to say. The index of improvement is the increase of strength. In reading the literature one finds cases in which creatine and creatinine have returned to normal. I know of no more complete studies than those of Harris and Brand; in talking with them I found that they were not decided as to whether the creatine or creatinine changed at all from the original levels after the intake of glycine. They were not enthusiastic about the chemical studies.

Whether larger doses would be more effective, we cannot say. Doses of 40 Gm. have been given to children with no better results than were obtained with 5 Gm. a day, and it must be remembered that glycine was so expensive earlier that it was difficult to get a sufficient amount for treatment in these cases.

As to combining ephedrine with glycine, I may refer to Boothby, who stated that in myasthenia gravis the strengthening action of glycine is increased by ephedrine. He suggested giving glycine first and then ephedrine. He thought that ephedrine may have some effect on the autonomic nervous system, and that this effect is augmented by feeding glycine. He believed that possibly the metabolic changes may be due to disturbance in the autonomic nervous system.

In regard to studies of the ductless glands in these patients, we made none; they were made in other clinics from which we received the patients. In the case of the child with Fröhlich's syndrome, roentgenologic studies showed no change in the sella turcica. Water and sugar metabolism, too, were apparently normal.

As to injections of pilocarpine and epinephrine, this had been carried out in our cases at other clinics. From those who gave the treatment I have the statement that the children who had been able to rise from a sitting position at the onset of treatment were unable to do so after 100 or 125 injections.

As to the psychology of treatment, I cannot say anything definite. The patients were all very young, and we did not notice any more enthusiasm in the children at the end of the treatment than at the beginning. They were accustomed to clinics; we were just "other doctors" to them, doing something else, and they did as we asked them.

CHOKED DISK AND PAPILLITIS: DIFFERENTIAL DIAGNOSIS BY THE PROTEIN CONTENT OF THE AQUEOUS. DR. ELIAS SELINGER (by invitation).

This article will be published in full in a later issue of the ARCHIVES.

PECULIARITIES OF THE INTRACRANIAL VENOUS SYSTEM AND THEIR CLINICAL SIGNIFICANCE. DR. PERCIVAL BAILEY.

It is important for the neurosurgeon to understand the peculiarities of the intracranial venous system. There are variations in the relations of the transverse and straight sinuses at the torcular Herophili. The left transverse sinus often seems to be a direct continuation of the straight sinus and the right transverse sinus, a continuation of the longitudinal sinus, with only small anastomoses. Under these conditions ligation of the left transverse sinus in a combined approach to a tumor of the acoustic organs is apt to result in serious disturbance of the internal circulation of the brain. It is well to scrutinize the torcular region in a good stereoscopic roentgenogram taken with the occiput on the plate before undertaking this operation.

The cerebral veins are divided into two groups, the internal and external veins, with inadequate anastomoses between them. The internal group drains almost exclusively through the great cerebral vein of Galen. Since this vein runs directly over the pineal body, it greatly increases the hazard of operation for a pineal tumor. It must be avoided at all cost, since its occlusion gives rise to serious symptoms, as Bagley has shown, such as coma with hyperpyrexia, elevation of pulse and respiratory rate, contraction of the pupils, rigidity of the limbs and exaggeration of the tendon reflexes. More chronic obstruction of this vein seems to cause a mild hydrocephalus.

It has long been known that the peculiar course of the superior cerebral veins renders them liable to injury, but it seems not to be sufficiently realized that ligation of the external cerebral veins is not an innocuous procedure. The superior cerebral veins may be rather evenly spaced along the longitudinal sinus, but in many cases, with the exception of a small frontal group, practically all of these veins enter the sinus in a group near the upper extremity of the central sulcus. In such a case ligation of these veins on one side, as is sometimes necessary when a meningioma develops in this region, causes a hemiplegia predominating in the leg which, although usually transitory, may be permanent. It has long been known, of course, that ligation of the superior longitudinal sinus behind the entrance of these veins causes paraplegia.

Ligation of other superficial veins may also cause permanent neurologic defects. There seem to radiate from the posterior extremity of the lateral fissure three systems of superficial veins: the rolandic veins, going roughly over the central gyri to the longitudinal sinus; the middle cerebral veins, running down the lateral sulcus to the sphenoparietal or cavernous sinus, and the lateral occipital veins, extending backward and downward to the transverse sinus. These three systems may anastomose freely with each other but often are independent, in which case ligation of the main trunks will cause symptoms. Ligation or injury of the middle cerebral trunk may be, at least in part, responsible for the facial palsy and epileptic attacks beginning in the throat and face which so often follow the lateral intradural approach to the pituitary region. If this is on the left side, aphasic symptoms may also result. On the left side aphasia may follow the ligation of the lateral occipital veins at their entrance into the transverse sinus. Hemianopia may result from ligation of the internal occipital vein during pineal operations (Cairns).

Another vein is the petrous, which drains the dentate nucleus of the cerebellum. This vein must be tied in the suboccipital approach to the fifth nerve. In every case in which I have ligated this vein there has been considerable cerebellar disturbance afterward, even though the cerebellum itself was not traumatized otherwise. The disturbance in every case has been transitory, although one patient who had been an iron worker was obliged to find other occupation because he no longer felt himself steady when working on high scaffolding.

SUICIDE: REPORT OF A FAMILY OF SUICIDES WITH HISTORY. DR. LOUIS B. SHAPIRO (by invitation).

Seven members of one family in four generations committed suicide, and another made several suicidal attempts. Two patients were regarded as definitely having mental disease, while the remaining five had never been considered as psychotic. Suicidal attempts may be due to temporary mental disorders, such as abortive attacks of manic-depressive psychosis. Manic-depressive psychoses occurred in three successive generations of this family, together with marked schizoid features in other members. A tendency to anticipation was shown in that mental disease appeared at an earlier age in each succeeding generation.

DISCUSSION

DR. MEYER SOLOMON: Heredity as a causative factor of suicide has been emphasized too much. It seems to me that unless there is conclusive, positive evidence with exclusion of all possible acquired causes one is not justified in assuming heredity, in the sense of mendelian units, as the cause. The subtle influences of environment and family situations are often overlooked. I have observed families in which there have been two or three suicides in two generations. The suicidal attempt of a son in one family, for example, could be traced to a similar attempt by his mother several years before, of which he had full knowledge. Imitation, therefore, is a factor of importance. Does Dr. Shapiro believe that in the study he has presented the hereditary tendency to suicide plays a dominant rôle?

DR. D. M. OLKON: Since Dr. Solomon emphatically denies that heredity has anything to do with suicidal tendencies, might not one ask him, first, by what specific knowledge he makes such denials and, second, how he establishes that environment alone is always the dominant factor? Is it not a common observation that suicides are few in the total population in any given environment, and is it not true that even in a given psychotic population the number of suicides is comparatively small?

That environment is a potent factor in many forms of behavior can be conjectured, but that heredity plays no part cannot be granted on logical grounds. Even if the knowledge of heredity is meager, as Dr. Solomon claims, it nevertheless shows many outstanding facts which hardly need emphasis; e. g., the resemblance of offspring to parents, familial resemblances and the nonreversion from one species into another among all animals indicate that such entities are doubtless carried into the zygote by the gametes. Moreover, if, as Morgan put it, a gene can carry definite entities which are reproduced in structure, can one logically deny entirely that the personality patterns are carried by genes or zygotes? Can it be definitely said that the personality make-up depends entirely on the environment, and that environment alone shapes the patterns of qualities of character, precipitates conduct and also determines self-destruction?

Dr. Shapiro has traced a family history in which many suicides occurred. The same family pedigree discloses that many members were considered to be psychotic. Some lived in state mental hospitals, and some, though outside of institutions, had mental disturbance. There were seven suicides in this lineage. Is it a valid assumption that each and every suicide in this group was caused by environment? One would have to assume that, in the popular vernacular, a sort of "environmental lightning" struck seven times in the same spot. It seems to me that in considering a history such as this one is forced to assume the opposite of Dr. Solomon's views; namely, that there were patterns in the mental make-up which made suicide a dominant tendency, and at a particular time when the environment stress was too exacting these patterns expressed themselves in execution.

DR. LOUIS B. SHAPIRO: In answer to Dr. Solomon's question as to whether I think that the tendency to suicide in this family was inherited, I do not wish to make a definite statement. As Dr. Solomon pointed out, the environment and

the heredity are too complex, and I think that from the study of this one group the question cannot be answered. I do believe that in many suicides of so-called normal persons there may be an inherited tendency to mental disease that has not been recognized.

Regular Meeting, March 15, 1934

JAMES C. HASSALL, M.D., *Vice President, in the Chair*

INJURIES TO THE VERTEBRAE AND INTERVERTEBRAL DISKS FOLLOWING LUMBAR PUNCTURE. DR. CHARLES N. PEASE (by invitation).

Following lumbar puncture many patients complain of pain and stiffness of the back. The persistence of these symptoms for several days is evidently due to more than trauma to the soft tissues by the introduction of the needle. It has long been known that the spinal puncture needle may be inserted too far into the canal, resulting in the clogging of the needle by the material from the intervertebral disk. Roentgen films in these cases reveal a variety of changes. In some there is narrowing of the intervertebral disk at the site of puncture; in others diminished intervertebral spaces and sclerosis of the vertebrae are seen, and in others there is a definite osteomyelitis of the bodies of the vertebrae.

Recently, investigation of this problem has been undertaken. Work on the cadaver demonstrates that in performing spinal puncture there is likelihood of pushing the needle into the intervertebral disk. There is no means of being sure whether the needle passed through the canal into the intervertebral disk.

The nucleus pulposus varies in consistency from semigelatinous to gelatinous. It is under pressure in the intervertebral fibrocartilage. When the spine is flexed for the performance of a puncture, there is increased pressure placed on the disk, thereby adding increased pressure on the nucleus pulposus. When the needle penetrates the disk, the fluid escapes into the lumen of the needle and through the perforation made by the needle. Narrowing of the intervertebral disk after such a procedure is demonstrated by roentgenograms.

If the spine is not completely flexed and the needle is introduced too far, the venous sinusoid on the posterior portion of the body of the vertebra may be punctured, resulting in flow of blood from the needle.

From the foregoing observations it may be concluded that the elements of the spinal column may be injured by lumbar puncture. These findings add another complication to the long list of accidents following spinal puncture.

DISCUSSION

DR. VICTOR E. GONDA: Were roentgenograms made of the spinal columns in some cases before the unfortunate spinal punctures were made? The last pictures impressed me with the thought that there may have been some pathologic change which was not previously recognized, and that this went on following the puncture.

DR. LEWIS J. POLLOCK: Was herniation of the nucleus pulposus frequent in adults? It is often found in persons presenting evidence of hypertrophic osteoporosis of the spine. I think that Schmorl has called attention to this.

DR. CHARLES N. PEASE: In none of the clinical cases, except the last, were roentgenograms taken before the puncture, but the patients did not have any symptoms referable to the back before the lumbar puncture.

In answer to Dr. Pollock, Schmorl reported herniation of the nucleus pulposus resembling one or two instances that I have shown. The herniations were into the bodies of the adjacent vertebrae from the flat surface of the disk. However, it is known that the nucleus pulposus can be extruded into canals, as in the cases reported by Dandy in which trauma ruptured the posterior wall of the intervertebral disk with the formation of a chondroma.

PAROSMIA IN TUMOROUS INVOLVEMENT OF THE OLFACTORY BULBS. DR. HARRY A. PASKIND.

This article will be published in full in a later issue of the ARCHIVES.

PHILADELPHIA NEUROLOGICAL SOCIETY

Feb. 23, 1934

D. J. MCCARTHY, M.D., *President, in the Chair*

A CASE OF AMYTAL COMPOUND (AMYTAL WITH AMIDOPYRINE) POISONING RESEMBLING ACUTE EPIDEMIC ENCEPHALITIS. DR. FREDERIC H. LEAVITT.

This case is of interest in that it represents a mistake in diagnosis of the etiologic factor concerned in the production of a syndrome resembling acute epidemic encephalitis. The patient, aged 14, her sister, aged 16, and the mother had an attack of influenza in the latter part of October 1933. Two weeks after the conclusion of this attack, the elder sister was extremely somnolent for a period of days; she could be aroused by vigorous shaking and would then drop back into profound sleep. When she was awakened it was discovered that she had definite extra-ocular weakness, producing a conscious diplopia, and left cerebellar symptoms together with irritative phenomena of the pyramidal tract affecting the left side of the body. The spinal fluid showed a slight increase in the lymphocyte count but no other abnormalities. This sister remained in a somnolent phase for several weeks, and organic signs persisted for many months. In the case of the younger sister, the psychologic fulcrum was a remark that I made in her presence to both parents to the effect that I thought it advisable for the mother and the older child to go to Florida for the rest of the winter, though it was not deemed essential to send the younger sister. I was surprised to be called a few days later by the family physician who told me that the younger child also apparently had acute encephalitis. He stated that a somnolent condition had developed from which it was impossible to arouse her and, owing to the fact that this child also had had influenza, it was thought that her condition as well as that of her sister was post-influenzal encephalitis. There was no fever, however, and none of the organic phenomena that were present in the sister could be detected in the younger child, other than a convergent squint which seemed to be constant. All tendon reflexes were completely abolished. The hands and feet were cyanotic, cold and sweaty. The pulse rate was extremely slow, between 40 and 60, and respirations were proportionately slow, being about 10 per minute. As it is rather unusual to find two cases of encephalitis in one family at the same time, I was not surprised to discover that the cause of the somnolence in the second child was an intentional overdose of an amytal compound. The mother discovered a capsule of this preperation in the child's bed, and when she examined the original bottle discovered that seventeen capsules were gone. The child remained in a somnolent condition for four days and then gradually emerged; by the end of the week she was normal. The patient then confessed that she had taken seventeen capsules, each of which contained 1½ grains (0.097 Gm.) of amytal, and 3 grains (0.195 Gm.) of amidopyrine. There was no suicidal intent, but the patient stated that she had taken the capsules because she wanted to go to Florida with her sister and she knew that the mother took the capsules to produce sleep, so she calculated that if she took enough she would go to sleep as her sister had done. Needless to say, the second child was not taken on the southern expedition. She rapidly recovered her mental activities, but for from two to three weeks following mental recovery the tendon reflexes remained completely abolished and she complained of persistent diplopia and presented visible evidence of extra-ocular unbalance. The extremities remained definitely cyanosed and cold for several weeks. I attributed these

vascular changes to the toxic effects of the amidopyrine rather than to the amytal. Six weeks later the child was well, the cyanosis had disappeared and the tendon reflexes were again normal.

A Case of Lead Poisoning from Gasoline Impregnated with Tetra-Ethyl Lead. Dr. Frederic H. Leavitt.

This case is reported as one of lead poisoning in industry. For many months the patient had worked as a mechanic in a repair garage, his particular duty being to wash the greasy metal parts of cars with gasoline. During this time he had washed his hands and arms from ten to fifteen times a day in lead-impregnated gasoline and also had used a hair brush soaked in gasoline to brush the grease and oil from other parts. The first symptom occurred five or six months after he started this work and was of the nature of intense abdominal "cramps." This was followed by persistent vomiting and obstinate constipation. At one time he went as long as ten days without a bowel movement; coincident with this episode he lost power in the extensor muscles of both wrists. At the same time he noticed weakness and some pain in the muscles of both arms and legs. Complete wrist drop developed, with wasting of the thenar and hypothenar groups of muscles of both hands. At no time was the patient conscious of any subjective sensory disturbances. He was admitted to the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases eight months after the onset of the paralysis; roentgenograms were taken of the long bones, which failed to show any deposits of lead at the epiphyseal lines. No decided anemia was present while he was at the hospital, nor was there stippling of the red cells. After several months of intensive treatment in the hospital there was no restoration of function in the paralyzed extensors of either wrist, and the atrophy was unimproved. The supinator longus muscle of each forearm remained unaffected.

The case is reported as an industrial accident to draw attention to the danger of workers using lead-impregnated gasoline without being advised of the danger in its use.

DISCUSSION

Dr. S. F. Gilpin: I saw a woman recently who took 18 grains (1.16 Gm.) of sodium amytal, the only result being twenty-four hours of sleep. I saw one patient who twice took 60 grains (3.9 Gm.) of barbital; she slept for two or three days and came out of the state with no trouble. I saw a man who, as nearly as could be determined from the history given, took about 260 grains (16.9 Gm.) of barbital; he was in bad condition from alcohol before taking the drug. It was impossible to revive him.

The case of lead poisoning impresses me with the carelessness of the patient. Every gasoline pump is distinctly marked that the gas is not to be used for washing hands or machinery. It is to be used for fuel only. I believe that such gas, and even the fumes are highly poisonous. A man who should know told me recently that during a vacation he was in a small garage working on his car and was made acutely ill from the fumes escaping from gasoline in an open container.

Dr. C. W. Burr: The real value of this case is to make one think in terms of prophylaxis. I am sure that workers in the factories where tetra-ethyl lead is manufactured are being protected and have learned what to do to prevent poisoning. This man, who worked around garages, should have been advised of the danger. I am sure there are many cases of this kind, and the number will increase until the workers are taught to take care of themselves. I suspect that the men with this particular type of lead poisoning who wash automobile parts in gasoline work with the fluid all the time; they could not become poisoned only from skin absorption, but must receive some by inhalation also. This man has fortunately escaped all mental symptoms.

Dr. R. A. Matthews: My associates and I at the Philadelphia Hospital for Mental Diseases have used sodium amytal in large quantities for several years and have found it satisfactory and efficacious. No untoward effects from the drug

have yet been noted; we learned by accident that morphine enhances the effect of the drug considerably. If morphine is administered when the effects of the sodium amytal are beginning to wear off, the length of the narcosis can sometimes be greatly prolonged. In patients in whom an ordinary dose of sodium amytal does not readily produce narcosis, the desired effect can often be obtained by the addition of a small dose of morphine. We have used the two drugs in combination not infrequently, and thus far no untoward effects or fatalities have occurred. It appears that the trouble arises when amytal (not sodium amytal) is used in combination with morphine. Amytal and amytal with amidopyrine are apparently much more toxic than sodium amytal. One patient received 27 grains (1.75 Gm.) of sodium amytal by mouth in a single dose. When the effects of the drug began to wear off after about twenty hours of sleep, she was given ¼ grain (16 mg.) of morphine sulphate, which produced narcosis for another twelve hours. She suffered no ill effects from the combination of the drugs. Did Dr. Leavitt's patient complain first of pain in the extremities? I know of a child who when given amytal with amidopyrine during an attack of whooping cough experienced rather severe pains in the legs which disappeared rapidly after the medication was discontinued.

DR. F. H. LEAVITT: When this girl was first seen she was so stuporous that she could not complain of pain; later she exhibited weakness and lethargy, and the latter condition persisted for two weeks. Both of these patients exhibited a chemical poisoning syndrome, which in the first simulated encephalitis, with complete recovery, and in the second produced intense neuritis and secondary atrophy, with no recovery of function despite many months of treatment.

AN UNUSUAL CASE OF CHRONIC SUBDURAL HEMATOMA. DR. STUART ROWE.

This case is reported because of the unusual diagnostic difficulties involved and because it illustrates the method of treatment for subdural hematoma now being used in the neurosurgical clinic of the Hospital of the University of Pennsylvania.

The patient, a boy, aged 13, was apparently well until Oct. 12, 1933 (about four months prior to admission), when in swinging over a small ravine on a high swing he fell approximately 40 feet. According to an observer, he struck the ground in a squatting position, his chin striking against his knees with considerable force. He was taken to a hospital where he remained unconscious for five days. There were three prominent symptoms: On returning to consciousness the patient complained of diplopia; when he began to move about and get out of bed a marked weakness of the right arm and a tendency to stagger to the right were noted, and for four weeks the patient continued to have severe generalized headaches at frequent intervals. Approximately four or five weeks after the accident two additional symptoms appeared: 1. The boy had the first of a series of three attacks of transient paralysis of the right side, preceded by jacksonian sensory phenomena. These attacks began with a sensation of numbness and tingling in the right foot, the peculiar feelings spreading rapidly up the right side of the body until the leg, arm and tongue were involved and also almost completely paralyzed. During the seizure speech was barely understandable and sounded "as though he had mush in his mouth." Apparently he had no difficulty in understanding correctly what was said to him. The attacks lasted from five to fifteen minutes and were not accompanied by loss of consciousness, incontinence, biting of the tongue or other features of generalized epilepsy. 2. At about the time of the onset of these seizures blurring and impairment of vision were reported by the boy, a symptom which became steadily more prominent until at the time of admission (Feb. 5, 1934) he was almost blind.

The patient was seen early in February by Dr. J. H. Dewey of Trenton, N. J., who referred him to the University Hospital on February 5. Examination showed an uncomfortable, depressed, rather emotional boy, who stated positively that he did not have any headache but wanted to be left alone. Examination of the cranial nerves gave negative results with the exception of the ocular changes. Vision was reduced to vague perception of hand movements and light; each optic disk was elevated 4 diopters and showed many hemorrhages and exudates; the pupils

were dilated, with the left slightly larger than the right, and reacted poorly to light. The only abnormality of motor power which could be detected was slight weakness of the movements of the right arm against resistance, particularly as compared with the left. Sensation seemed normal throughout, and no astereognosis was present. The tendon reflexes were all greatly depressed but obtainable with reenforcement, the right achilles jerk being more active than the left. A suggestion of a Babinski reflex occurred on plantar stimulation of the right foot. Careful testing for cerebellar dysfunction revealed only a mild rebound phenomenon in the right arm, with some awkwardness of the finger movements of the same hand, findings which may well have been due to weakness.

Roentgenographic examination of the skull showed evidence of rather long continued high intracranial pressure, including widening of the sutures, increase in the size of the pituitary fossa and atrophy of the dorsum sellae. It was the opinion of the roentgenologists that the changes seen could not have been produced by an intracranial lesion present for only four months.

While it was obvious that the patient had an intracranial lesion, there was some uncertainty as to its exact nature and location. It was thought by some that the high degree of intracranial pressure, the roentgenographic changes in the skull and the marked loss of vision favored strongly a diagnosis of tumor while others believed that the apparent close relation of the whole illness to the severe head trauma pointed to a chronic subdural hematoma. The neurologic evidence and the history suggested that the lesion was in the left frontal region, and ventriculography was performed to confirm or disprove this.

The marked shift of the entire ventricular system to the left and the thinning out and downward displacement of the frontal horn of the right lateral ventricle seen in the films after ventriculography on February 7 showed clearly that the boy had a lesion in the right frontal area. Dr. Frazier immediately began craniotomy, but after making one trephine opening in the right frontal region decided to open the dura mater to investigate what lay beneath. When the thickened dura mater was nicked there was a sharp spurt of cerebrospinal fluid tinged with fresh blood, and in the course of a few minutes 40 or 50 cc. escaped. It could then be seen that the cortex in this area was covered with the greenish membrane characteristic of subdural hematoma. A second perforation, about 5 cm. posterior to the first, showed the same membrane, but a third perforation in the parietal region revealed only normal cortex. Two trephine openings were made on the left side of the head opposite those in the right frontal region, but no evidence of subdural hematoma was found, and the cortex seen appeared normal. The wounds were closed with a small rubber tissue drain left beneath the scalp near the first perforation. A considerable amount of slightly blood-tinged spinal fluid drained for approximately forty-eight hours, when the drain was removed and the wounds healed rapidly.

After a rather stormy immediate postoperative course of a few days, with complete blindness, severe headaches and some fever, the patient convalesced uneventfully. At present he is bright and alert, has normal motor power and is regaining some vision. He distinguishes hand movements readily, although only in relatively small temporal fields. He has 3 diopters of choked disk in each eye. There has been no evidence of recurrence of a collection of fluid or of progression of the neurologic disturbance.

DISCUSSION

DR. M. J. COOPER: Dr. Rowe's excellent presentation of this case has covered the essential features. I wish to emphasize the clinical picture at the time when I examined the patient on admission to the hospital. The high degree of choking of each optic disk, with great impairment of vision, the dilated and practically nonreactive pupil on the left side and practically normal pupil on the right and the history of transient motor and sensory attacks affecting the limbs of the right side, with mild objective weakness of the upper part of the right limb and reflex hyperactivity on the right at the time of examination, all pointed toward a cerebral lesion on the left side. Kaplan (*Brain* **54**:430 [Dec.] 1931) recently reiterated the

importance of the dilated pupil as a localizing sign in cases of chronic subdural hematoma and called attention to the occurrence of bilateral hematoma. Mydriatics had not been used in this case prior to the examination. The findings, considered in the light of the history, seemed to present the classic picture of traumatic hematoma, with localization on the left side, probably in the frontal region.

The demonstration of the hematoma on the right side in this case, the apparent absence of any such lesion on the left side and the roentgenologic evidence of increased intracranial pressure antedating the injury raise the question of a lesion existing, independently of the trauma. It is conceivable that this suspected preexisting lesion also produced the pupillary changes and the motor and sensory attacks affecting the right limbs. Considering the case in retrospect, it now seems to me that such severe trauma to the head as was suffered by this patient might be expected to cause focal hemorrhages and areas of softening scattered throughout the cerebral tissue, in addition to the demonstrated subdural hematoma, and that the signs pointing toward focal involvement of the left hemisphere might be accounted for by the effect of such softenings and scar formation. It will be interesting to learn of any further developments in this case.

DR. STUART ROWE: In regard to Dr. Cooper's thought about the possibility of a traumatic lesion existing on the left side, I think that it is possible that the boy has such a condition. If he continues to have choked disk for a longer time than my associates and I expect, following the release of the pressure we shall probably make an encephalogram in order to see whether there is any coexisting intracranial lesion.

BILATERAL GANGLIOGLIOMA OF THE FRONTAL LOBE WITH EPILEPSIA PROCURSIVA. DR. JAMES W. WATTS.

F. K., entered the Hospital of the University of Pennsylvania on Sept. 18, 1933, in Dr. Spiller's service, with the chief complaint of convulsions. There was no family history of epilepsy or of nervous or mental disease. The past history threw no light on the etiology of the attacks. The menses began at the age of 13 years and have been regular. At 14 months mastoidectomy was done for mastoiditis followed by erysipelas. Since, the patient has had an intermittent discharge from the right ear.

The present illness began seven years ago, at the age of 13, when she began to have "running attacks." While walking down the street, for no apparent reason she would suddenly break into a run and run for several hundred feet before stopping. The running was entirely involuntary and on realizing what she had done the patient would feel very foolish. Once she ran across the street in the midst of heavy traffic, but was not injured. About nine or ten months after the onset she was stopped during one of the "running attacks" by the mother. She immediately became unconscious. During the second year of the illness, convulsions appeared for the first time. An aura, consisting of an indescribable sensation in the epigastrium, is followed by a generalized convulsive attack with unconsciousness, followed by a period of drowsiness. Occasionally there are incontinence of urine during an attack and headache and vomiting following it. These seizures occurred as often as several times a week and sometimes as seldom as once in three months.

In April 1933, turning of the head and eyes to the right preceded the generalized convulsions. In the middle of September 1933, the patient had three severe convulsions associated with frontal headache and vomiting.

A roentgenogram of the skull made at the Hahnemann Hospital showed a calcareous deposit in the midline in the frontal region.

On examination there was discharge from the right ear, and the scar of an old mastoidectomy was seen. The audiometer indicated 74.3 per cent loss of hearing in the right ear, and 35.2 per cent loss in the left ear. The remainder of the physical and neurologic examination gave normal results. There was no evidence of choked disk, nor was a defect found in the visual fields. Roentgenograms of

the skull showed a thin shell of calcification in the midline in the frontal region which was interpreted by Dr. Pancoast as being calcification within the capsule of a tumor. The patient was transferred to Dr. Frazier's service, and because there was no evidence of increased intracranial pressure or signs of a focal character revealed by the examination or by the nature of the convulsion an encephalogram was made. There was good filling of the ventricular system, which was distorted in the region of the anterior horns by a large calcified lesion in the midline which tended to flatten the anterior horns, displacing the ventricular system posteriorly. It was noted that there was little or no air in the midline or in the frontal region.

With the patient under avertin anesthesia, supplemented by procaine hydrochloride, Dr. Frazier turned down a right transfrontal osteoplastic flap. A dural flap was reflected with the base toward the longitudinal sinus. A large cyst, 4 or 5 cm. in diameter, adjacent to the falx and extending anteriorly nearly to the frontal pole, was disclosed. The pia-arachnoid forming the wall was thick and opaque. At first it was thought that this cyst might have caused the deformity noted in the encephalogram.

However, when the cyst was opened and the right cerebral hemisphere was retracted laterally, a large solid tumor lying on the floor of the anterior fossa was exposed. It extended under the falx cerebri, a part of it lying under each frontal lobe. The tumor was readily separated from the normal brain. It had a slightly reddish appearance and was firm on palpation. It was unlike anything previously seen in this clinic.

With the Bovie unit, a specimen was taken for biopsy. In the frozen section it had an appearance not unlike that of a granuloma. As it did not resemble tuberculoma or gumma grossly, it was decided to proceed with the excision. This was carried out by removing the tumor, bit by bit, with the electrosurgical unit. By this method relatively little bleeding was encountered. After about three fourths of the tumor had been removed, the systolic blood pressure fell to 50 mm. of mercury. A blood transfusion was started, the wound was carefully closed, and the removal of the remainder of the tumor was postponed for a second operation.

On further study, Dr. Alpers was of the opinion that the tumor should be classified as a ganglioglioma. It was quite vascular, and in places there was a perivascular infiltrate composed chiefly of lymphocytes. A thick connective tissue capsule surrounded the tumor and sent numerous fibrous tissue strands into it. Most of the neoplastic cells had round or oval vesicular nuclei with coarse chromatin structure and no nucleoli, evidently astrocytes. Lobulated nuclei and multinucleated cells were numerous. Among these cells were not a few with moderate and large vesicular nuclei with large, prominent nucleoli. When visible, the cytoplasm trailed off into a swollen apical dendrite. No definite Nissl substance was found in cells which looked like ganglion cells. No mitotic figures were seen. In some parts of the tumor fibrils ran in close connection with the cells, running in many directions. In other parts the fibrils were grouped in bundles.

The patient's convalescence was uneventful. Since her discharge from the hospital on Dec. 5, 1933, she has had only two attacks. The first, apparently grand mal, occurred in bed, with clonic movements of the extremities. The second was an attack of forced running ending in a brief period of unconsciousness.

This case fits in well with the descriptions of epilepsia procursiva. Based on the location of the tumor in the case which has just been described, the suggestion is made that the pathologic process of epilepsia procursiva lies chiefly or entirely within the prefrontal region.

DISCUSSION

DR. B. J. ALPERS: This tumor was extremely interesting from the histologic standpoint. It proved to be a ganglioglioma which was present between the frontal lobes, arising probably at the base of the brain near the chiasm. It contained many immature ganglion cells and many cells of glial origin as well. By virtue of its location as well as its structure it could be readily classed as a ganglioglioma.

With regard to the running movements, are these not expressions of subcortical, probably thalamic, activity, rather than of cortical action, and do they not correspond to the running movements observed in thalamic animals?

DR. E. A. SPIEGEL: The first to observe running movements was Nothnagel, who injected toxins into the corpus striatum. He was really the first to find symptoms from injuries to the caudate nucleus. This tumor is localized in the midline at the base of the frontal lobe. The head of the caudate nucleus comes close to the cortex. The possibility should be borne in mind that the running movements in this case were due not to the frontal lobe but to the caudate nucleus.

DR. R. H. THOMPSON: This case presents many features of interest. First there was difficulty in determining whether the condition was organic epilepsy, the result of a large tumor of the frontal lobe, or so-called idiopathic epilepsy. The history shows that there may be no difference between the clinical manifestations of epilepsy resulting from organic disease of the nervous system and those of the idiopathic type.

Many phases have been noted which are characteristic of ordinary epilepsy, i. e., the onset at puberty, the procursive or running attacks with disturbed consciousness and confusion, the sleep walking episode, likely a nocturnal manifestation of epilepsia procursiva, and finally the typical grand mal seizures. In the absence of any localizing loss or irritation of cortical function all these factors tend toward a diagnosis of epilepsy without organic cause.

Jacksonian attacks indicating the location of a tumor of the brain may begin with a local sensory or motor aura involving some one part of the body. Progressively other parts may become involved, and if the irritation passes the midline the seizure becomes generalized and consciousness is lost.

In this instance the tumor was external to the cortex, was situated in the midline on the floor of the anterior fossa and was large and calcareous. Whatever irritation to the motor cortex might occur would, therefore, be bilateral and equal and symmetrical on the two sides. In other words, localized seizures could not be expected unless certain parts of the cortex were more vulnerable than others. The emotional exciting factor accordingly would precipitate generalized convulsions, although the attacks would still be localizing and of the jacksonian type. It is suggested, therefore, that the bilaterality of the tumor produced symmetrical irritation in each motor cortex with a resulting bilateral jacksonian hemiclonic phenomenon and unconsciousness. Such a conception aids in maintaining a distinction between convulsions as a result of traumatic irritation and convulsions due to metabolic dyscrasia or some other unknown factor.

In this case there is added evidence that it is possible for a cerebral tumor to give rise to generalized convulsions for many years. Pritchard reported five cases of cerebral tumor as a cause of generalized epileptic attacks of long standing. One patient had a large subcortical glioma in the left occipital and posterior parietal lobes with a history of epileptic seizures for eighteen years before any signs indicating a local cerebral lesion were detected (*Lancet* **2**:842 [Oct. 17] 1931).

Dr. Spiller had a man, aged 28, under his care in 1932 who had had generalized epileptic seizures for thirteen years, and in whom a diagnosis of tumor of the left temporal lobe was made. Dr. Frazier, on operation, found and evacuated a large cyst of the left temporal lobe. The history and neurologic examination in Dr. Spiller's service, however, gave evidence of the nature and locality of the growth.

In the case under discussion the diagnosis of tumor of the brain could not have been made but for the roentgenographic investigations. An added instance is thus afforded of the value of such examinations in general convulsive seizures, and it is recommended that similar procedures are warranted in all cases of so-called idiopathic epilepsy.

DR. N. W. WINKELMAN: Is it not true that most of the ganglioneuromas that have been reported in the literature have been in the temporal rather than in the frontal lobe?

DR. B. J. ALPERS: It is true that many gangliogliomas are found in the temporal lobe, but they are found also at the base of the brain in the medulla, the third ventricle and the region of the midbrain.

DR. J. W. WATTS: I am afraid that I cannot answer these questions. I think that Dr. Thompson was right in emphasizing the need for a careful complete study of all epileptic patients. Concerning Dr. Alper's remarks, I do not think it necessarily follows that because running movements may occur after removal of the frontal lobes forced running could not have its origin there. A stimulus arising in the frontal region might spread posteriorly to the premotor and motor areas, with resulting movements of the extremities. Penfield's observations after cortical stimulation indicate that the neuronal discharge spreads from one part of the brain to adjoining areas. It seems to me that a stimulus arising in the frontal region might initiate the "forced running" described in this case.

TREATMENT OF SPASMODIC TORTICOLLIS WITH SPECIAL REFERENCE TO PSYCHOTHERAPY, WITH REPORT OF A CASE. DR. JOSEPH C. YASKIN.

From a review of the literature it appears that there are few cases in which spasmodic torticollis has been proved to be due to organic disease of the brain. These cases were associated principally with encephalitis or torsion spasm, and the torticollis formed merely a part of the clinical picture. In some of these cases surgical intervention is reported to have been of benefit. In the majority, the organic origin of spasmodic torticollis has been unproved. Nevertheless, a number of the patients were benefited by removal of foci of infection, elimination of reflex causes and surgical intervention.

It appears that most of the cases in which organic disease cannot be proved are, at least in part, psychogenic. If this is so, every patient with spasmodic torticollis should receive the benefit of psychotherapy before surgical intervention is attempted. The older forms of treatment, which depended on gymnastics, reeducation, rationalization and ordinary suggestion, did not reach the fundamental causes and were responsible for many failures. Modern psychotherapy, which attempts to reach the roots of the conflicts of which the hyperkinesia is a symbolic expression, may succeed. In the case reported, faulty psychosexual development with varying anxiety was the fundamental cause of the spasmodic torticollis, and even partial resolution of the underlying conflict brought about a temporary recovery. Sodium amytal was used to obtain a rapid rapport with the patient.

An objection frequently raised against psychotherapy is that it is tedious and time-consuming. The same objection may be raised in the treatment of all psychoneuroses. Psychotherapy, on the other hand, attempts to treat the underlying causes and may often succeed when surgical intervention fails. Certainly the surgical treatment is not uniformly successful. At the same time, one should not dogmatically state that surgical intervention has no place in psychogenically produced cases. One may strongly stress that when definite organic causes are not demonstrated the patient should receive a careful psychiatric investigation and an adequate course of psychotherapy.

DISCUSSION

DR. E. A. SPIEGEL: Dr. Yaskin has given a careful study and analysis of the conditions which lead to torticollis. A number of years ago I was interested in the question of producing torticollis experimentally by combining central lesions with injection of toxins. Contrary to the experience in man, lesions of the extrapyramidal centers were not followed by hypertonicity even in monkeys. I thought that it might be possible to secure the desired result by combining such lesions with the injection of different toxins. Bulbocapnine, in a dose of 1/20 grain (0.003 Gm.), produces catalepsy in a cat. The limbs and the back are flexed, and the animal can be put in any posture desired. Analysis of this condition shows that the subcortical ganglia have something to do with this condition. I thought that it would be interesting to study changes in tonicity after lesions of the fore-

brain by injecting this toxin. Such changes were found even in lesions of the motor cortex. The motor cortex was destroyed in a normal cat on one side; the animal showed no definite change in posture and tonicity and no torticollis. A few weeks after this operation, catalepsy was produced by the administration of bulbocapnine and the head was rotated toward the side opposite the cortical lesion. If one tried to move the head into its normal position a strong resistance could be felt; in other words, the combined effect of the central lesion and intoxication induced a posture similar to torticollis in a human being.

UNILATERAL AMYOTROPHY: ITS DIAGNOSTIC IMPORTANCE FOR CEREBRAL LOCALIZATION. DR. A. SILVERSTEIN and DR. N. WINKELMAN.

This article will be published in full elsewhere.

Book Reviews

Estructura y sistematización de los gliomas y paragliomas. By Pió del Rio-Hortega. Archivos españoles de oncología, volume 2, no. 3 and 4, February 1932.

The author, in a monograph of two hundred and fifty pages, discusses the literature critically, classifies gliomas and paragliomas on a histogenic basis and gives a detailed description of the various types. He believes that the work will be of value in that the descriptive material is based entirely on preparations made with a single technic, that in which silver carbonate and its various modifications are employed. He defines glioma and paraglioma, stating that the neuroglial network of the nervous system only, exclusive of the meninges, endoneurium and perineurium, is truly neuroglial and capable of forming gliomas. He excepts the cells of Schwann, which he considers equivalent to oligodendroglia and which are therefore, theoretically at least, capable of forming peripheral gliomas. He includes under the term paraglioma tumors formed by neuroblasts and ganglion cells and those derived from the germinal cells of the epiphysis and from the epithelium of the choroid plexus, which, although offering some traits in common with the gliomas, should be studied under a different category.

He regrets the prolixity of names founded on inaccurate morphologic and embryonic concepts and the multiplicity of neoplastic types that are not well characterized. He praises the work of Bailey and Cushing, his main points of difference being that he does not think that they have been able to establish the existence of the medulloblast and that in their classification there is a mixing of tumors of nervous origin with others of neuroglial and pineal origins. He disagrees with Schaffer's definition of an undifferentiated adendritic cell. He favors a histogenic classification, disagreeing with Roussy and Oberling, whose classification he criticizes for complicating an already obscure picture. He criticizes the tendency of many investigators to create many minor types. Under embryologic data he stresses histogenesis as being the common ground for description. He believes that the medulloblasts of Bailey and Cushing, as well as the undifferentiated cells of Schaffer, are in reality the progenitors of the oligodendroglia, capable of developing into astrocytes but not into nerve elements. He gives a detailed description of the embryologic development of the cells which go to form gliomas and paragliomas.

In regard to systematization, the primitive medullary epithelium has four evolutional possibilities: the formation of glioblasts, neuroblasts, pineoblasts and choroidoblasts—the elements going into the formation of the four fundamental types of brain tumors. The glioblasts are tripotential; if they migrate, they form astroblasts and oligodendroblasts, and if they remain in situ they form the ependymal glio-epithelium. The structure of the gliomas is based on these three neuroglial types and on their typical or atypical evolutionary forms. A diagram is given showing the medullary epithelium as the origin of the glioblast, neuroblast and pineoblast; and the glioblast, in its turn, of the astroblast, oligodendroblast and ependymal glio-epithelium. Gliomas are formed by neuroglial elements in their embryonic, transitional and adult forms and can be divided into five classes: glioblastoma, astroblastoma, astrocytoma, oligodendrocytoma and glio-epithelioma. Theoretically, all varieties corresponding to the evolutional stages of each type of cell are possible, but only some have an accurate delineation, others being so similar that their differentiation is extremely difficult. The names are of limited value, often corresponding to the predominating element.

Gliomas are formed from the proliferation of embryonic cells resting in the nerve tissue as: (*a*) cells arrested under the medullary epithelium; (*b*) cells detached from the neuro-epithelium, but keeping intact their embryonic characters,

and (*c*) heterotropic invaginations of the neuro-epithelium resulting in inclusions in the nerve tissue. All these cells of interrupted evolution keep their embryonic capacity of atypical proliferation and further differentiation. The glioblastomas in all their forms are rich in cells resulting from the proliferation of elements with little or no capacity for differentiation. The astroblastomas, oligodendrocytomas and astrocytomas, which are relatively poor in cells, come from elements arrested at an incomplete state of differentiation and are endowed with an insignificant proliferative capacity but with a great tendency toward morphologic and structural differentiation. Two types of differentiation may take place: (1) when all the cells develop at the same time and in the same measure, resulting in isomorphous tumors, and (2) when they develop in stages, giving rise to heteromorphous tumors, in which may be associated glioblasts, astroblasts, astrocytes and oligodendrocytes.

The most embryonic tumors have indifferent elements (primitive glioblasts) with a tendency to conserve epithelial characters (strata of cells, circular systems), to lose these characters and be scattered in a disorderly way or to form perivascular systems. Regression to the epithelial type and progression to the neuroglial type may coexist in the same tumor. When the glioblastic differentiation begins, the proliferative faculty of the cells diminishes and the protoplasm emits contrapolar expansions, with a tendency to seek out vessels and form groups about their sheaths. This angiotropism varies greatly and leads to the formation of histologic varieties.

The second stage of the glioblastic evolution (secondary glioblast) is characterized by an increase in protoplasm and the appearance in it of delicate expansions with greater tendency to form vascular attachments. Some of these glioblasts are almost purely protoplasmic, while others show fibrillary differentiation of the protoplasm without adopting astroblastic forms.

The morphologic differentiation of the cells corresponds to the astroblastic type. They increase in volume, acquire new expansions and show monopolarity of the protoplasm, which gives out one long expansion near the nucleus. Secondary expansions appear elsewhere about the cell.

Complete maturity is shown by fibrous and protoplasmic astrocytic forms which, however, may present atypical appearances.

The neoplastic oligodendroblast passes through a primary phase, in which the protoplasm is scanty and only indications of expansions are seen, to the oligodendrocytic form with more protoplasm, which emits a variable number of expansions that show occasional T-shaped branchings. Apart from this type, variations corresponding to the first and second types of oligodendroglia are seen. The oligodendrocyte as seen in tumors is not a fully differentiated cell, its complete evolution being frustrated by the absence of nerve fibers.

All gliomas formed of immature cells, morphologically little differentiated, may be included in the glioblastomas. These have a very embryonic form corresponding to the medulloblast or indifferent cell and a more differentiated form which is difficult to separate from the astroblast. There are two fundamental types of glioblastoma—isomorphous and heteromorphous—between which exist numerous varieties, depending on the degree of typical or atypical cellular evolution and on the part played in the architecture by vasculoconnective tissue.

In the isomorphous glioblastomas, the cells are found in a fairly uniform morphologic phase, while the structure varies considerably. The term isomorphous does not signify an absolute equality of structure, but a tendency of the cells to develop closely to the normal fashion. In the last analysis, the cellular types are reduced to : (1) those retaining some epithelial characters, (2) those attracted by vessels forming gliovascular systems, (3) those repelled by vessels forming orographic structures and (4) those showing funicular and lobular arrangements due to the participation of vasculoconnective tissue. The heteromorphous glioblastomas are characterized by: a lack of equilibrium and proportion in the nuclear cytoplasmic ratio, the manner of cellular evolution, their proliferative rhythm and their grouping in masses, their vascular development, and their

tropisms. It seems as though, in an embryonic glioblastoma, the cells had grown in a disorderly fashion, with great powers of expansion and infiltration. Their chief characteristic is cellular pleomorphism based on nuclear abnormalities and monstrosities and on luxurious proliferation. The participation of vessels and connective tissue and the presence of multiple necrotic foci give a special character to these blastomas.

The astroblastomas, representing the forms of passage of the embryonic gliomas to the adult form, are not as characteristic in appearance as the latter. There is no boundary line between glioblastomas and astroblastomas. There are astroblastomas both rich and poor in cells, showing a limited differentiation in regard to the cytoplasmic expansions, of which one, longer and thicker, has the greatest importance. A great many gliomatous astroblasts are monopolar and bipolar, with a preponderance of pyriform and cometary types; but multipolar types with rarely dividing branches are often seen. These tumors show few signs of proliferative activity.

Astrocytomas are polymorphous and may have zones of protoplasmic elements and other zones of fibrous elements, with the corresponding morphologic and structural transitions. Protoplasmic astrocytomas have neurologia cells with numerous expansions which are lax, ramified, interlaced into plexuses and without a great tendency to attach themselves to vessels, although they often show typical vascular feet. Fibrous astrocytomas have cells with extremely long and fibrous expansions, which give diverse histologic aspects of lax or compact structure: (1) small cells having isomorphous characters and moderate fibrillary differentiation, (2) many extremely fibrous cells, (3) with cells and fibrous structures extremely compact, and (4) showing a great abundance of fibers associated in fascicles.

Oligodendrocytomas are tumors formed principally of oligodendrocytes, but it is rare to find one that offers that single type of cell, and there may exist in them zones with an abundance of astroblasts and astrocytes. The cells have a typical morphology, but all forms are not present, as the nerve fibers necessary for their typical configuration are lacking. The cellular characteristic is polymorphism, but no equilibrium in the distribution of expansions exists. There are subvarieties of oligodendrogliomas on the basis of the preponderance of small cells, large cells or extremely lengthy schwannoidian elements. The last are found in tumors of the optic nerve. The encephalic oligodendrocytomas have cells with few expansions, rarely dividing and with a great polymorphism. The oligodendrocytomas of the optic nerve are formed by bipolar, tripolar and multipolar elements, the bipolar elements being most abundant, with a very atypical evolution in the schwannoidian sense. To these are added others with a feeble protoplasmic evolution, to which may be attributed the formation of mucoid substances.

Glio-epitheliomas are formed from the remains of the ependymal glio-epithelium and may show typical or atypical characters. The adjective glio-epithelial refers only to cells having a tendency to form on surfaces or to form cords without losing their ependymal character, for if this disappears and if there are vascular systems present, the tumor becomes a glioblastoma. The glial character may disappear completely and be replaced by epithelial characters. Two cytologic forms may be seen—embryonic and adult—corresponding to the character of the epithelium of the encephalic cavities before and after primitive evolution. When the cells keep their glio-epithelial character, ependymogliomas are formed, which are characterized by the rootlike expansions of the cells. If this character disappears, the cells become more epithelial, and ependymo-epitheliomas are formed, in which the elements form little islands around neuroglia and canals or vesicles which on the surface lend the tumor an alveolar appearance.

The paragliomas (neuroblastomas and neurocytomas) are formed from rests of embryonic corpuscles or from adult neurocytes that are but slightly differentiated morphologically, such as the bipolar cells of the retina. The neuroblastic elements sometimes perpetuate their original characters and at other times initiate typical

or atypical adult forms and may come to acquire complete morphologic and structural differentiation. In the first case, neuroblastomas are formed, and in the second, neurocytomas. These tumors may show: (1) undifferentiated cells with unipolar or bipolar protoplasm without neurofibrillary differentiation or a tendency to form neurites; (2) cells with an initial neurofibrillary differentiation and a clear nucleus containing a large nucleolus; (3) cells with more developed cytoplasm, with dendrites and a typical ganglion cell nucleus, and (4) cells with greater somatic development and numerous dendrites showing complete neurofibrillary differentiation, Nissl bodies and axonal expansion. There are, then, tumors of immature elements, or neuroblastomas, and others with mature ganglion cells, or neurocytomas.

Neuroblastomas are preferentially retinal and show a varied structure corresponding to the cellular evolution, the atypical forms present, the concomitant regressive phenomena and the participation in their structure of neuroglial and connective tissue elements. From the evolutionary standpoint there are five types: (1) that with very embryonic elements, tending to show epithelial arrangements and forming true rosettes; (2) that with almost undifferentiated elements, in which the epithelial tendency is less but the neuroblastic character is almost lacking; (3) that with neuroblasts having an epithelial appearance, but with nuclear differentiation not accompanied by cytoplasmic development; (4) that with elements, usually bipolar, having the nucleus well differentiated, and showing neurofibrillary differentiation of the cytoplasm, and (5) that with multipolar neuroblasts in transition to ganglion cells, without axonal expansions or complete structural differentiation.

Neurocytomas are tumors formed of typical or atypical ganglion cells, but those which are made up exclusively of these cells are rare. They frequently present phenomena of regression and signs of amitotic division of the nucleus. The cells are associated with typical incompletely differentiated neurocytes, neuroblasts and gliocytes.

Pineoblastomas and pineocytomas are tumors of the epiphyseal group having characters similar to those of the embryonic pineal gland, and it is possible that they have completely developed pineal cells. The pineoblast does not correspond to the initial phase of organization of the epiphysis, but to a stage part way toward the complete organization. The greater part of the germinal elements keep their embryonic characters, which are similar to those of the glioblast, while a small part develop into neuroglial elements. The fundamental characters of the pineoblastoma rest: in the specific cells, which show few expansions and which rarely end in clubs; in the interstitial neuroglial cells disseminated among the pineoblasts, and in the vasculoconnective stroma that causes the lobular arrangement.

The choroid epithelioma corresponds to an atypical and exuberant growth of the papillary tufts of the choroid plexus, being a connective tissue substratum with an epithelial investment.

Macroglia: There are sometimes in gliomas reactional phenomena of local macroglia, with the formation of perineoplastic and intraneoplastic gliosis, and at other times gliosis is seen at a distance from the neoformation, which seems to depend on the phenomena of local and general compression.

Microglia: These cells do not react to the excitants that cause the violent reproduction of the embryonic neuroglia cells, and when they do increase the cause must be attributed to phagocytic stimulants. There are few microglia in gliomas, and these carry out their respective functions of phagocytosis, during which they lose their complicated ramifications, become thicker and globose and end by forming compound granular corpuscles loaded with fat.

Child Guidance Clinics: A Quarter Century of Development. By George S. Stevenson, M.D., and Geddes Smith. Price, $1.50. Pp. 186. New York: The Commonwealth Fund, 1934.

The development of the child guidance clinic within the twenty-five years which have elapsed since the founding of the Chicago Juvenile Psychopathic Institute is

reviewed by Stevenson and Smith from both a historical and a descriptive point of view. The first clinic established under the auspices of the National Committee for Mental Hygiene—at St. Louis, in 1922—is described; unfortunately, the authors fail to name the personnel of this pioneer agency. The Dallas clinic, established in the following year, introduced two significant novelties: It marked the first expansion of the field to include problems of childhood not associated with delinquency, and it was the first to be underwritten by a community chest or public funds. The last demonstration clinic was set up in the favorable soil of Philadelphia—favorable because of the long-established neuropsychiatric facilities (the Pennsylvania Hospital, the Graduate School of the University of Pennsylvania, the Philadelphia Neurological Society and other institutions and organizations)—and was the first to provide a technic for the training of graduate students in psychiatry. In reviewing the history of the demonstration clinics, the authors stress the lessons which they taught, particularly the importance of adequate and independent financial backing, the dangers of municipal control and the desirability of avoiding too close a connection with the juvenile court.

In their description of the operation of the clinic, the authors describe the routine of accepting and presenting a case. They suggest that cases of epilepsy, feeblemindedness, psychosis and gross organic neurologic disorder be excluded. The tendency to overemphasize psychic factors to the neglect of the determining sociologic and economic forces is deplored. As Stevenson and Smith view it, the clinic may lean toward one of two extremes in its policy: (1) It may consider itself a responsible social institution able to utilize many assets of the community not available to the unaided patient; accordingly, it would initiate and direct procedures and accept full responsibility for treatment. (2) It may endeavor to establish a significant personal relationship between the worker and the patient and use this rapport for the presentation of facilities, leaving to the family the active choice. In either event, the authors fear, treatment will have to be on superficial psychic levels. Intensive, deep therapy is not usually expedient and, when used, "its results are sometimes futile, sometimes embarrassing, and not too often successful."

For the treatment of faulty habits in children, reliance must be placed on the physician or pediatrician, rather than on the clinic. That pediatricians too seldom have a psychiatric point of view is realized, however, and this deficiency is to be laid at the door of the medical school. To some extent this gap may be filled, if chiefs in pediatrics in hospitals would transmit something of an attitude of mental hygiene to the residents in this specialty. To train students in pediatrics by presenting mental hygiene as an "outside" subject is, the authors suggest, an error. Juvenile psychiatry should be woven closely into the pattern of clinical pediatrics.

Both the administrative management and the medical examination in the clinic should be assigned to the psychiatrist. Too often the latter takes the duty of medical examinations casually. The medical examination offers the psychiatrist a valuable opportunity of cementing his relationship with the patient—an opportunity too often neglected. Justification of entrusting to the psychiatrist the administrative management of the agency is found in the fact that training in medicine with psychiatric emphasis is more likely than any other professional training to bring to the clinic the leadership which will assure a constant orientation to the whole organism.

Before setting up a child guidance clinic the sponsors should assure themselves that the community has adequate facilities for managing cases of the grosser deviations (epilepsy, defectiveness and psychosis); otherwise, these cases will swamp the new clinic and impede its services. It is also necessary to surround the clinic with adequate services for children (placing agencies, schools and other facilities); otherwise, the agency will have inadequate tools. Support should come from welfare federations, community chests, universities or well established endowments, rather than from taxes or school funds.

The book presents a somewhat sketchy but interesting history of the child guidance clinic movement. Procedures are indicated rather than described. It will be of service to an agency contemplating the setting up of a new clinic and to all workers in the movement. As a manual of practice it is inadequate, however, and most of the concrete suggestions offered consist of warnings as to what to avoid rather than instructions as to what to do.

Les tumeurs de la loge cérébelleuse. By T. de Martel and J. Guillaume. Price, 90 francs. Pp. 445, plus index, with illustrations. Paris: Gaston Doin & Cie, 1934.

This monograph is a systematic review of the symptomatology, pathology and surgical treatment of tumors of the posterior fossa, based on experience with one hundred and thirty-nine cases. While there is not much new material in the book, aside from the histories of cases, conclusions from the literature are carefully criticized point by point on the basis of the authors' experience. They call attention to the importance of vomiting as an early localizing symptom of tumors of the posterior part of the fourth ventricle and the prognostic significance of this manifestation. Vomiting may also be produced by irritation of the anterior part of the third ventricle. The early abolishment of the pupillary reflex may also be taken as a localizing sign in this instance of involvement of the corpora quadrigemina. Mental disturbances, especially confusion and depressive stages, may result from increased intracranial pressure. An excellent review is given of the question of forced attitudes. There is an interesting observation in regard to the place of origin of "tonic fits." During the course of an operation a series of these was initiated by section of the anterior extremity of the vermis with the electric needle.

The authors call attention to the value of Barré's plumb line test and palpation of muscle groups, with the patient in the erect position, in the demonstration of abnormalities of posture and of hypotonia. The presence of deafness, especially bilateral deafness, with tinnitus in the presence of a choked disk, may be taken to suggest involvement of the cochlear nuclei either directly or by an obstruction of the aqueduct of Sylvius. In a case of obstruction of the aqueduct by a very small tumor it was demonstrated that clonus and spasticity appeared only during periods of acute hypertension.

The section on pathology is well written, but contains little that is new. The various anatomic varieties of tumor are considered separately, as is the question of arachnoiditis. Lumbar puncture was performed in only three cases, but it had been performed in eighteen others. In two cases of medulloblastoma an increase in the number of cells was found, the number reaching one hundred and fifty in one case. In most instances there was an increase in protein. Ventriculography is highly recommended in doubtful cases, and the authors state that they have never had a serious accident resulting from it.

A novel surgical technic is recommended for exposure of the posterior fossa. The operation is performed in three parts. In the first stage the foramen magnum is enlarged and the arch of the atlas removed through an incision in the midline. The second part of the operation consists in forming an inverted horseshoe-shaped flap of skin and muscle between the tips of the mastoid at the external occipital protuberance. The third part of the operation consists in preparing a flap of bone extending above the lateral sinuses and lateral to the mastoids by means of de Martel's circular saw. The muscles are then turned downward, attached to the flap of bone. From this point the operation is continued according to the usual technic of Cushing. At the close of the operation a drain is placed in the fourth ventricle, and the flap is sutured in place with wire. The authors believe that postoperative hyperthermia can be adequately treated by ventricular puncture. They advise against the use of radiotherapy without preliminary decompression. The operative results are approximately the same as those obtained in most specialized clinics. No patient with medulloblastoma survived more than fifteen months. An unusual number of tuberculomas are reported, amounting to 7 per cent of the whole. Seven of ten patients with tuberculoma were apparently cured.

The monograph is clearly written and fairly well illustrated and will prove a valuable reference book in any neurologic clinic.

Mental Hygiene in the Community. By Clara Bassett, Consultant in Psychiatric Social Work, Division on Community Clinics, the National Committee for Mental Hygiene, Inc. Price, $3.50. Pp. 394. New York: The Macmillan Company, 1934.

A great deal of propaganda regarding the field of mental hygiene has been appearing in printed form. There has been little available to different professions or persons in the community that would give them some definite idea as to what mental hygiene means in their particular work or activity. This book has the purpose of filling that gap, and it takes up thoroughly details concerning mental hygiene as related to many fields of activity. It is worth while listing the headings of the chapters to show from what aspects mental hygiene is discussed: 1. Introduction; 2, Mental Hygiene and Medicine; 3, Mental Hygiene and Nursing; 4, Mental Hygiene and Social Service Angencies; 5, Mental Hygiene, Delinquency and Law; 6, Mental Hygiene and Parental Education; 7, Mental Hygiene and the Pre-School Child; 8, Mental Hygiene, Education and Teacher Training; 9, Mental Hygiene, the Church and Theological Training; 10, Mental Hygiene and Industry; 11, Mental Hygiene and Recreation; 12, Mental Hygiene and Psychiatric Institutions and Agencies; Bibliography, and Index.

Chapters 2 and 3 are very good. Chapter 5 contains good criticism of the lack of mental hygiene and of social sense in the training in the legal profession. Chapter 6 points out the need of training children in school for family relationships. Chapter 8 is excellent. Invaluable as possibilities for reference are the suggestions and questions given at the end of each chapter that furnish the groundwork for study and knowledge of the field of mental hygiene in relation to the contents of the chapter. A short bibliography is given, and the index is good.

As a whole, the book contains too much detail, and one chapter reads a great deal like another. Nevertheless, it is an excellent résumé of the relations of mental hygiene and its use in community service and will be valuable to all persons with community interests. It stresses the origin of many modern agencies and community functions. Many authorities of different points of view are quoted in relation to the discussion on topics relative to work in which such authorities have demonstrated good results. Each chapter may be considered a useful treatise by persons interested in the particular field discussed. The author is a woman who has been active in an organization that knows the mental hygiene field. She gives a hopeful sound to mental hygiene work and in this publication implies much that mental hygiene may do for various aspects of community life.

The Brain and Its Mechanism. The Rede Lecture Delivered Before the University of Cambridge, Dec. 5, 1933. By Sir Charles Sherrington. Paper. Price, 50 cents. Pp. 35. New York: The Macmillan Company, 1933.

The author's understanding of nerve physiology gives this small work an intensely interesting point of view in philosophic and psychologic speculations concerning the mind. He sees the function of the brain as that of a motor mechanism, and emphasizes the idea that life is an act, not a thought, but that refraining from an act is no less an act than the act itself, because inhibition is a nervous activity coequal with excitation. Excitation and inhibition cooperate and at any moment settle what the motor outcome will be.

Differences in the stimuli change the result, adapting the nodal point to a new stimulus as it happens with the simplest reflexes, for the same stimulus can from time to time call forth different results, sometimes amounting to actual reversal. The brain is a great field of convergence for signals. "The brain has thus arisen where signaling is busiest and is fraught most with a germ of futurity." In this complex nerve-net, signals started at different inputs meet and react together. The brain differs from the ordinary network because it can connect fresh

messages with an outgoing order. The new messages need only be repeated closely a number of times to become significant and acquire executive power. They amplify the animal behavior. The new connection improves with use. Under disuse it disappears. These new connections can develop without limit in man.

Sherrington accepts the view that as with the muscular acts of behavior run mental events so with the signaling in the brain-nets managing muscles runs mental action. The signals entering the brain are not mental, nor the excitant signals, but signaling which travels certain ways in the brain seems to acquire mental existence. Sherrington sees nothing different in the nerve-nets of the brain and states: "All is as elsewhere except greater complexity." He has great faith in common sense pressed for time, accepting and acting on acceptance.

The mental processes are not of the reflex type. They are through-line events in the brain between input and output. The history of the brain suggests that mind is, at the outset, a sort of adjunct to the nerve management of motor behavior. For the mind loses touch with the suite of motor behavior exactly at the point where the brain's executive can influence behavior no longer.

Sherrington regards the relation of mind to brain not merely as still unsolved but devoid of basis for its very beginning. But, undiscouraged, he sees at least one virtue in the problem: that to journey is better than to arrive, although comfort assumes arrival.

Les troubles du sommeil: Hypersomnies, insomnies et parasomnies. By Henri Roger. Price, 20 francs. Pp. 206. Paris: Masson & Cie, 1932.

Roger's small volume gives a survey of the field of sleep and its disorders. While insomnia has been a topic of discussion since the time of Job, other disturbances of sleep have come into prominence only relatively recently. Narcolepsy, recognized by Gelineau in 1880, the epidemic of encephalitis after the war, and experiments on the infundibular region have added much to knowledge of this indispensable function. Roger considers first normal sleep and the various theories as to its cause. The three principal ones are those concerned with the vegetative system, the cerebrospinal system and the psychic system, none of which is entirely conclusive, but all of which present important conclusions. In the chapter on insomnia or hyposomnia, which is naturally long, the symptomatology, pathogenesis and treatment are discussed at length. The author describes in detail the various therapeutic methods, not only drugs but also baths, electricity and psychotherapy. In his opinion, the treatment of insomnia is far more complex than a few doses of barbital. The chapter on hypersomnia is more extensive than one would at first suppose, since many other diseases besides epidemic encephalitis may be characterized by pathologic somnolence. Narcolepsy receives detailed consideration and a number of the current theories are discussed; the use of ephedrine, however, is mentioned only on the basis of the preliminary report of Doyle and Daniels. Under the heading of parasomnias the discussion becomes more psychoanalytic; the author discusses dreams, nightmare, nocturnal terrors and somnambulism, although certain other phenomena related to disturbances of the function of sleep are also discussed.

The volume is written in an easy, direct style, condensed and clear, but not superficial, and should prove of value to the physician who is faced with the necessity of treating patients with disorders of sleep.

A Laboratory Manual of Neuro-Anatomy. By C. L. Davis, M.D., and H. S. Rubinstein, B.S., M.D. **Part II. Stereographic Plates.** Price, $3. 30 plates. Baltimore: William Wood & Company, 1933.

This valuable series of stereograms deserves high praise because for the student they are a satisfactory substitute for the actual specimens which are usually obtainable only in a laboratory. With the exception of the first three plates, the rest are entirely original. They merit all the praise that can be given to such an effort.